Available online at www.sciencedirect.com
SCIENCE DIRECT®
Science of the Total Environment 326 (2004) 1–31
Science of the Total Environment
An International Journal for Scientific Research into the Environment and its Relationship with Humankind
www.elsevier.com/locate/scitotenv

Review

Review of selenium toxicity in the aquatic food chain

Steven J. Hamilton*

US Geological Survey, Columbia Environmental Research Center, 31247 436th Avenue, Biological Resources Division, Yankton, SD 57078-6364, USA

Received 25 August 2003; accepted 13 January 2004

Abstract

In many environmental contaminant situations selenium has become the primary element of concern because of its bioaccumulative nature in food webs. Initial concerns about selenium were related to fish kills at Belews Lake, NC, Martin Lake, TX, and Kesterson Reservoir, CA, and to bird deformities at Kesterson Reservoir. Additional concerns were identified under the National Irrigation Water Quality Program at Salton Sea, CA, Kendrick, WY, Stewart Lake, UT, and Grand Valley and Uncompahgre Valley, CO. Recent studies have raised concerns about selenium impacts on aquatic resources in Southeastern Idaho and British Columbia. The growing discomfort among the scientific community with a waterborne criterion has lead the US Environment Protection Agency to consider a tissue-based criterion for selenium. Some aquatic ecosystems have been slow to recover from selenium contamination episodes. In recent years, non-governmental researchers have been proposing relatively high selenium thresholds in diet and tissue relative to those proposed by governmental researchers. This difference in opinions is due in part to the selection of datasets and caveats in selecting scientific literature. In spite of the growing selenium literature, there are needs for additional research on neglected organisms. This review also discusses the interaction of selenium with other elements, inconsistent effects of selenium on survival and growth of fish, and differences in depuration rates and sensitivity among species.

Keywords: Selenium; Food chain; Interactions; Species sensitivity; Thresholds; Controversy

1. Introduction

Selenium is an essential micronutrient in animals (Klasing, 1998; Eisler, 2000). It has three levels of biological activity: (1) trace concentrations are required for normal growth and development; (2) moderate concentrations can be stored and homeostatic functions maintained; and (3) elevated concentrations can result in toxic effects. Industrial and agricultural activity has hastened the release of selenium from geologic sources and made them available to fish and wildlife in aquatic and terrestrial ecosystems around the globe. Agricultural drain water, sewage sludge, fly ash from coal-fired power plants, oil refineries, and mining

*Tel.: +1-605-665-9217; fax: +1-605-665-9335.
E-mail address: steve_hamilton@usgs.gov (S.J. Hamilton).

0048-9697/04/$ - see front matter © 2004 Elsevier B.V. All rights reserved.
doi:10.1016/j.scitotenv.2004.01.019

of phosphates and metal ores are all sources of selenium contamination of the aquatic environment (Heinz, 1996; Eisler, 2000; Lemly, 2002a).

Uptake of selenium by biota can be from water or diet. Uptake of water-soluble selenium by fish and wildlife can be either by gills, epidermis or gut. However, dietary exposure of animals to selenium is usually the dominant pathway of uptake because animals are typically at higher trophic levels in the aquatic and terrestrial food webs (Dallinger et al., 1987). Several papers have documented and reviewed selenium in aquatic food chains from various viewpoints, such as those of an aquatic toxicologist (Saiki 1986), wildlife toxicologist (Ohlendorf, 1989; Skorupa, 1998), an ecologist (Maier et al., 1987), a research chemist (Presser, 1994; Presser et al., 1994), a modeler (Bowie et al., 1996), a national selenium expert (Lemly, 1999b), and other experts (Davis et al., 1988; Sorensen, 1991; Hamilton, 1998).

Bioaccumulation of trace elements in food chain components such as aquatic plants and aquatic invertebrates and the resulting effects on fish has been documented in aquatic ecosystems contaminated with mixtures of elements. However, in many of the reported situations, selenium has surfaced as the element of primary concern because of its propensity to bioaccumulate within the base of food webs: from water and sediment to aquatic plants and aquatic invertebrates (Cherry and Guthrie, 1977; Furr et al., 1979). Bioaccumulation of selenium leading to toxicological impact and change in aquatic communities has been intensively investigated in lab and field studies. Sorensen (1988) states, 'Fish kills [at Belews Lake, NC, and Martin Lake, TX] were considered a direct result of selenium release into the main basin of the lakes because several hundred analyses for metals, metalloids, physiochemical parameters, and pesticides provided essentially negative results except for sufficiently high levels of selenium in the water (approx. 5 μg/l) to warrant concern'. Lemly (1985) reviewed information in 10 studies of potential causes for disappearance of several fish species in Belews Lake, and of the 16 inorganic elements of concern, only selenium was present at elevated concentrations in water and fish. Saiki and Lowe (1987) measured several inorganic and organic chemicals in water and biota collected from Kesterson Reservoir area, CA, and concluded only selenium was elevated sufficiently to be of concern to fisheries resources. Nakamoto and Hassler (1992) measured 20 trace elements in fish from the Merced River and Salt Slough in the San Joaquin Valley, CA, which was primarily irrigation return flows, and concluded only selenium was present at toxic concentrations. Gillespie and Baumann (1986) concluded that selenium was the element causing the deformities and reduced survival of larval bluegills (*Lepomis macrochirus*) and not other elements (arsenic, cadmium, copper, lead, mercury, zinc) present in females from Hyco Reservoir, NC. Bryson et al. (1984) concluded that selenium was the only element elevated sufficiently in zooplankton collected from Hyco Reservoir, NC, and not other elements (arsenic, cadmium, copper, mercury, or zinc) to cause the 97% mortality of juvenile bluegill after 1 week of dietary exposure. Montgomery Watson (MW, 1999) concluded that selenium was the major element of concern associated with phosphate mining activities in the Blackfoot River watershed of Southeastern Idaho and not other elements (cadmium, manganese, nickel, vanadium and zinc). Hamilton et al. (2001a,b) measured several inorganic elements in various ecosystem components at three sites in the upper Colorado River during a reproduction study with endangered razorback sucker (*Xyrauchen texanus*) and concluded that selenium was the primary element of concern. Skorupa (1998) reviewed 12 environmental case studies of confirmed or highly probable selenium poisoning in nature, where other inorganic elements were present, but not of major concern.

This review covers aspects of selenium toxicity in the aquatic food chain that have not been usually addressed in other selenium reviews such as the emerging selenium contamination associated with phosphate mining in Southeast Idaho, infrequently cited selenium studies at Sweitzer Lake, CO, selenium interactions with other elements linked with delayed mortality in fish, inconsistent effects of selenium on survival and growth of fish, differences in depuration rates and sensitivity among species, ecosystem recovery from selenium con-

tamination, and controversy among proposed selenium thresholds.

2. Early selenium studies

Three independent studies of selenium toxicity published in the early 1980s foreshadowed the intensive selenium investigations that were began in the late 1980s and have continued today. Early dietary studies with selenite and rainbow trout (*Oncorhynchus mykiss*) were published in the early 1980s by Canadian researchers (Hilton et al., 1980; Hodson et al., 1980; Hilton et al., 1982; Hilton and Hodson, 1983; Hodson and Hilton, 1983; Hicks et al., 1984). These early selenium studies explored dietary requirements, elimination and uptake rates from water and diet sources, minimum dietary requirement for rainbow trout (between 0.15 and 0.38 μg/g in dry feed) for maximal storage, half-life period, influence of dietary carbohydrate, and toxic concentrations in water and diet. It was shown that plasma glutathione peroxidase homeostasis was maintained at up to 1.25 μg/g dry feed activity; toxicity occurred at 13 μg/g dry feed, but the authors speculated that dietary concentrations in excess of 3 μg/g in dry feed over long time periods might be toxic; liver and kidney were the primary tissues for storage; and excess dietary carbohydrate enhanced dietary selenium toxicity in rainbow trout. Similar responses to dietary selenium in terms of deficiency, requirements, and toxicity have been reported for fish, birds and mammals (Puls, 1994; Eisler, 2000).

A second series of investigations of a massive disappearance of several fish species in Belews Lake, NC, which received effluent from ash ponds of a coal-fired power plant, in the late 1970s was linked to selenium toxicity in the food chain (Cumbie and Van Horn, 1978; Holland, 1979; Lemly, 1982, 1985; Sorensen et al., 1984). Selenium concentrations in Belews Lake were approximately 10 μg/l in the main lake and approximately 5 μg/l in one arm where adverse effects were measured in fish, but there was little information on food chain organisms. Later studies examining the recovery of Belews Lake and its fauna were conducted by Lemly (1993b, 1997a, 2002b). The Belews Lake investigations led to additional studies at Hyco Reservoir, NC, another coal-fired power plant cooling reservoir with selenium contamination (Bryson et al., 1984; Woock, 1984; Bryson et al., 1985; Finley, 1985; Woock et al., 1987; Coughlan and Velte, 1989). Although other inorganic elements were elevated in water in these two lakes, selenium was the element of concern due to its bioaccumulation in the food chain and its toxicity to fish.

A third series of studies were related to fish die-offs in Sweitzer Lake in Western Colorado and field investigations in Colorado and Wyoming (Barnhart, 1957; Birkner, 1978). These two studies concluded selenium toxicity was occurring in fish via the food chain. Barnhart (1957) was the first publication to suggest that selenium in the food chain was causing fishery problems in Sweitzer Lake in Western Colorado. These studies in turn prompted two early investigations of dietary selenite toxicity to rainbow trout conducted by the Colorado Division of Wildlife (Goettl and Davies, 1977, 1978). Goettl and Davies (1978) reported that dietary selenium toxicity occurred between 5 and 10 μg/g dry diet, which is remarkably close to that reported by the Hilton/Hodson group and also to later dietary studies conducted with selenomethionine and other fish species in the late 1980s.

It is interesting to note that the publications of the Hilton/Hodson group and field investigations in Belews Lake, NC and Western Colorado did not mention each other's selenium investigations that were occurring during the same time period. The next location where a selenium contamination problem with a fishery was reported was at Martin Lake, TX, another cooling reservoir for a coal-fired power plant (Kirkpatrick, 1980; Garrett and Inman, 1984). Several selenium investigations were conducted, primarily using histopathology assessments with redear sunfish (*Lepomis microlophus*) and green sunfish (*Lepomis cyanellus*) (Sorensen et al., 1982, 1983; Sorensen and Bauer, 1983, 1984; Sorensen, 1986, 1988). These studies reported elevated selenium accumulation in muscle, hepatopancreas, liver and kidney, lower condition factor, and cytotoxicity in erythrocytes, hepatopancreas, kidney, liver and ovary.

Table 1
Selenium concentrations in fish exposed to selenium in the diet or water and adverse effects

Exposure route, species, and weight (g)	Selenium exposure concentration (diet: μg/g; water: μg/l)	Selenium form	Exposure period (day)	Whole-body selenium (μg/g)	Effect	Reference
Diet						
Rainbow trout						
79	9	Selenite[a]	294	NG[b]	Mortality	Goettl and Davies, 1978
1.3	13	Selenite[c]	80	5.2[d]	Mortality, growth	Hilton et al., 1980
0.6	11–12	Selenite[c]	112	4.0–4.5[e]	Kidney, weight	Hilton and Hodson, 1983
Chinook salmon						
4.2	26[f]	SLD[g]	34	8.4[f]	Migration	Hamilton et al., 1986
~1	9.6	SLD[g]	90	6.5	Mortality	Hamilton et al., 1990
	9.6	SEM[h]	90	5.4	Mortality	
~1	5.3	SLD[g]	90	4.0	Growth	Hamilton et al., 1990
	18.2	SEM[h]	90	10.8	Growth	
Fathead minnow						
0.12	20	Mix[i]	56	5.4	Growth	Ogle and Knight, 1989
0.0001	55–70	Rotifer[j]	7–9	43–61	Growth	Bennett et al., 1986
Striped bass						
251	39	Fish[k]	80	15[f,l]	Mortality	Coughlan and Velte, 1989
Bluegill						
≈0.3	45	Zooplankton[m]	7	25	Mortality	Bryson et al., 1984
2.8	54	Mayfly[n]	44	31[f,l]	Mortality	Finley, 1985
Adult	13	SEM[h]	260	NG	Reproduction	Woock et al., 1987
0.2	6.5	SEM[h]	59	4.3[o]	Mortality	Cleveland et al., 1993
≈2.4	5.1[p]	SEM[h]	180	5.5	Mortality	Lemly, 1993a
Adult	33	SEM[h]	140	19	Reproduction	Coyle et al., 1993
Razorback sucker						
0.005	2.4–5.1	Zooplankton[q]	30	3.6–8.7	Mortality	Hamilton et al., 1996
Water						
Rainbow trout						
0.08	47	Selenite	60	5.2[f]	Mortality, length	Hunn et al., 1987
Chinook salmon						
0.3	69	Mix[r]	60	3.8	Mortality	Hamilton et al., 1986 Hamilton and Wiedmeyer, 1990
Chinook salmon						
0.3	143	Mix[s]	60	4.9	Growth	Hamilton et al., 1986 Hamilton and Wiedmeyer, 1990
Chinook salmon						
0.3	67	Mix[t]	60	4.5	Mortality, growth	Hamilton et al., 1986 Hamilton and Wiedmeyer, 1990
Razorback sucker						

Table 1 *(Continued)*

Exposure route, species, and weight (g)	Selenium exposure concentration (diet: μg/g; water: μg/l)	Selenium form	Exposure period (day)	Whole-body selenium (μg/g)	Effect	Reference
~0.1	472	Mix[u]	90	5.9	Growth	Hamilton et al., 2000a
Bonytail						
~0.1	236	Mix[v]	90	9.4	Growth	Hamilton et al., 2000a

[a] Selenite: selenite incorporated in standard Colorado trout diet.
[b] NG: not given.
[c] Selenite: selenite incorporated in a casin-torula yeast trout diet.
[d] Derived from figure 2 in Hilton et al. (1980)
[e] Carcass.
[f] Reported as wet weight and converted to dry weight assuming 75% moisture.
[g] SLD: western mosquitofish (*G. affinis*) collected from San Luis Drain, CA, used as fish meal portion in an Oregon moist pellet diet.
[h] SEM: selenomethionine incorporated into fish food.
[i] Mix: 25% selenomethionine, 25% selenate, and 50% selenite incorporated in fish food.
[j] Rotifer: rotifers fed selenium-laden algae.
[k] Fish: red shiners (*N. lutrensis*) sieved weekly from Belews Lake, NC, where they were chronically exposed to 10 μg/l selenium and food-chain selenium under natural conditions.
[l] Muscle tissue.
[m] Zooplankton: zooplankton collected from Hyco Reservoir, NC.
[n] Mayfly: burrowing mayfly nymphs (*Hexagenia limbata*) collected from Belews Lake, NC.
[o] Derived from figure 3 in Cleveland et al. (1993).
[p] Exposure included waterborne exposure to 4.8 μg/l selenium.
[q] Zooplankton: zooplankton collected from Sheppard Bottom ponds 1, 3, and 4 at Ouray NWR, UT.
[r] Mix: 3023 μg/l boron, 96 μg/l molybdenum, 69 μg/l selenium, and water simulating the San Joaquin River, CA.
[s] Mix: 6046 μg/l boron, 193 μg/l molybdenum, 143 μg/l selenium, and water simulating the San Joaquin River, CA.
[t] Mix: 2692 μg/l boron, 92 μg/l molybdenum, 67 μg/l selenium, and well water at Yankton, SD.
[u] Mix: 16 μmg/l arsenic, 5040 μg/l boron, 80 μg/l copper, 40 μg/l molybdenum, 408 μg/l selenate, 64 μg/l selenite, 264 μg/l uranium, 16 μg/l vanadium, 160 μg/l zinc, and water simulating the Green River, UT.
[v] Mix: 8 μg/l arsenic, 2,520 μg/l boron, 40 μg/l copper, 20 μg/l molybdenum, 204 μg/l selenate, 32 μg/l selenite, 132 μg/l uranium, 8 μg/l vanadium, 80 μg/l zinc, and water simulating the Green River, UT.

3. Later selenium studies

Although the primary interest was on ameliorating mercury toxicity, a series of experiments in lakes in Sweden also tested the interactive effects of selenium and mercury on the aquatic community (Paulsson and Lundbergh, 1989, 1991, 1994). These studies confirmed that selenium readily reduced mercury accumulation in fish, but selenium bioaccumulated in fish via the food chain if waterborne selenium concentrations were greater than 3–5 μg/l. Fish kills of yellow perch (*Perca flavescens*) occurred in four of the 11 lakes in their study, and concern was raised that selenium might have been linked to the fish kills, but they could not exclude it as a cause. Using a precautionary approach, Lindqvist et al. (1991) recommended against the use of the selenium amelioration technique in mercury-contaminated lakes in Sweden. Similar findings were reported in a series of studies by Rudd, Turner and others (Rudd et al., 1980; Turner and Rudd, 1983; Turner and Swick, 1983) who also investigated the ability of selenium to ameliorate the toxic effects on fish inhabiting a mercury-contaminated lake in the English–Wabigoon River system in Ontario, Canada. They cautioned that selenium amelioration of mercury should be approached with caution because selenium readily and efficiently accumulated in the food organisms and fish, especially through the food chain, and recommended that selenium additions be limited to 1 μg/l.

In contrast to the fisheries-inspired studies discussed above, adverse effects in wildlife, especially water birds, was the major impetus behind selenium investigations begun in the mid 1980s at Kesterson Reservoir located adjacent to Kesterson National Wildlife Refuge (NWR) in the San Joaquin Valley of California (USFWS, 1990). Irrigation of seleniferous soils in the San Joaquin Valley resulted in high selenium drain water that was transported by the San Luis Drain to a series of evaporative ponds constituting Kesterson Reservoir. The most dramatic discovery of the Kesterson investigations were deformed embryos of water birds such as black-necked stilts (*Himantopus mexicanus*), American coots (*Fulica americana*), and various duck species (Ohlendorf et al., 1986a; Hoffman et al., 1988). The Kesterson investigation was perhaps the most extensive examination of a selenium contaminant problem and resulted in over a hundred reports and publications. Numerous studies of selenium effects on ducks were conducted in the laboratory (e.g. Hoffman et al., 1992a,b; Heinz and Fitzgerald, 1993a,b; Heinz et al., 1996) and the field (e.g. Ohlendorf and Skorupa, 1989; Ohlendorf et al., 1990). Additional studies were conducted with reptiles and amphibians (Ohlendorf et al., 1988; Ohlendorf, 1989), and mammals (e.g. Clark, 1987; Paveglio and Clifton, 1988; Clark et al., 1989). Similar selenium-induced reproductive failure in water birds occurred in the Tulare Basin of California (Skorupa and Ohlendorf, 1991).

Fish disappearances also occurred at Kesterson NWR (Harris, 1986; Vencil, 1986). Concerns about potential effects on fish lead to several fish studies, primarily with diet, but also with water borne exposures, conducted in the laboratory (e.g. Hamilton et al., 1986, 1990; Hamilton and Wiedmeyer, 1990; Coyle et al., 1993; Cleveland et al., 1993), and in field studies (e.g. Saiki and Schmitt, 1985; Saiki, 1986; Saiki and Lowe, 1987). Additional studies were conducted with invertebrates (e.g. Ingersoll et al., 1990; Maier and Knight, 1991; Maier et al., 1993; Hansen et al., 1993; Malchow et al., 1995) and microcosms (Besser et al., 1989, 1993).

The selenium contamination problem at Kesterson Reservoir inspired newspaper reporters to travel throughout the Western states investigating other irrigation projects for selenium problems. The end result was a series of news articles titled 'Selenium-toxic trace element threatens the west, the [Sacramento] Bee uncovers conspiracy of silence' (Harris et al., 1985). The reaction of the US Department of the Interior (DOI) was to establishment of the National Irrigation Water Quality Program (NIWQP; Feltz and Engberg, 1994). This program assessed concentrations of selenium and other elements at 26 sites in 17 Western states to determine if irrigation-related problems existed at DOI constructed or managed irrigation projects, national wildlife refuges, or other wetland areas for which the DOI has responsibilities under the Migratory Bird Treaty Act, the Endangered Species

Act, or other legislation. The program has produced hundreds of reports and publications that have exponentially increased the selenium literature and provided the foundation for several review articles (e.g. Engberg and Sylvester, 1993; Seiler, 1995, 1996; Naftz, 1996; Engberg, 1998, 1999; Skorupa, 1998).

Although most of the studies in the NIWQP were of a contaminant survey nature with few biological effects studies, the NIWQP has drawn on other biological effects studies in the selenium literature and produced guidelines for interpretating residues of selenium and other elements of concern (Skorupa et al., 1996; USDOI, 1998).

4. Recent selenium studies

Although Kesterson Reservoir has been closed and capped, selenium loading in the Central Valley of California, the San Joaquin River, the San Joaquin-Sacramento Delta, and San Francisco Bay (Bay) still occurs. Consequently, selenium impacts on biota in the Bay have been a concern since the early days of the Kesterson investigations (BISF, 1986) and continue to the present. In addition, oil refineries on the Bay release substantial amounts of selenium-contaminated waste water to the Bay (Johnson et al., 2000). Early studies in the Bay documented elevated selenium in various aquatic components (e.g. Ohlendorf et al., 1986b; Cutter, 1989). Several publications have linked selenium loading of the benthic food web in the Bay with potential adverse effects in diving ducks (e.g. Greenberg and Kopec, 1986; Ohlendorf et al., 1986b; Luoma and Linville, 1996; Hothem et al., 1998; Linville et al., 2002). One remediation effort to remove selenium from oil refinery effluent entering the Bay has been the use of artificial or constructed wetland at the Chevron Refinery (e.g. CH2M Hill, 1995; Hansen et al., 1998; Terry and Zayed, 1998). Selenium loading from oil refineries has been reduced from 11 to 15 pounds per day in 1986 to 1992 to 3 pounds per day in 1999 (Luoma and Presser, 2000). The wetland remediation technique for selenium immobilization has met with some criticism because of ecological risks in the wetland environment (Lemly, 1999b; Ohlendorf and Gala, 2000; Lemly and Ohlendorf, 2002). Selenium was a concern in a constructed wetland in the Albuquerque, NM area because selenium bioaccumulated to elevated concentrations in aquatic invertebrates, whereas concentrations of aluminum, arsenic, mercury, and silver did not (Nelson et al., 2000).

Research efforts in the San Francisco Bay have lead to substantial advances in the understanding of selenium cycling, especially food chain transfer of selenium and selenium loading of the Bay (Luoma and Presser, 2000). The Bay is characterized by a enhanced biogeochemical transformations to bioavailable particulate selenium and efficient uptake by bivalves and the predators such as surf scoter (*Melanitta perspicillata*), greater scaup (*Aythya marila*), lesser scaup (*Aythya affinis*), and white sturgeon (*Acipenser transmontanus*). Luoma and Presser (2000) have identified bivalves as the most sensitive indicator of selenium contamination in the Bay and developed a forecast model of selenium loading in the Bay based on various scenarios including input from the Sacramento and San Joaquin rivers, a possible extension of the San Luis Drain, oil refineries, and other sources.

Concerns for the recovery of endangered fish in the Colorado River have been expressed since the 1960s and efforts for their recovery began in earnest in the 1980s (USFWS, 1987). Information from the NIWQP in the late 1980s reported elevated selenium in water, sediment, and biota in the Colorado River basin. This information in turn raised selenium-related concerns about the impacts on fish and wildlife from agricultural irrigation of seleniferous areas in the upper Colorado River basin (reviewed in Hamilton, 1998). Studies with endangered razorback sucker revealed that selenium readily bioaccumulated in adults and eggs, increased deformities in larvae, and selenium-laden food chain organisms reduced larvae survival (Hamilton et al., 2001a,b). Hazard assessments of these and other studies revealed high hazards to razorback sucker in both the Green River in Utah (Hamilton et al., 2000b) and the upper Colorado River in Colorado (Hamilton et al., 2002a). A review of historical information for both currently endangered fish and the selenium literature led to the hypothesis that historical selenium contamina-

tion in the 1890 to 1910 period caused the decline of native fish inhabiting big rivers such as the Colorado pikeminnow (*Ptychocheilus lucius*), razorback sucker, and possibly others such as bonytail (*Gila elegans*) in the 1910 to 1920 period in the upper basin and in the 1925 to 1935 period in the lower basin (Hamilton, 1999). The NIWQP has undertaken remediation efforts at Stewart Lake on the Green River in Utah and the Gunnison and upper Colorado rivers in Colorado to reduce selenium loading, and in turn, reduce possible effects of selenium on endangered fish (Engberg, 1992; Butler, 2001; Darnall et al., 2002). Piping of lateral irrigation pipes in the Gunnison Valley, CO, has been reported to reduce selenium loading, especially of groundwater by 28% (Butler, 2001), and flushing of a large backwater channel near Grand Junction, CO, has substantially reduced selenium concentrations in water, sediment, aquatic invertebrate, and forage fish of piscivores (Hamilton et al., 2003).

An emerging selenium contaminant issue is developing in southeastern Idaho in streams draining areas with phosphate mining activities. The Phosphoria Formation covers a vast area encompassing areas of Idaho, Montana, Utah, and Wyoming. The mining process moves large amounts of waste materials away from the ore deposits and places them in dumps and landfills often located in valleys. Livestock grazing on vegetation growing on some seleniferous waste materials have been euthanized due to selenium toxicosis and others found dead from selenium poisoning (Caribou County Sun, 1999). Leaching of selenium and other elements from mined areas and waste materials has been reported (Desborough et al., 1999), which has resulted in elevated concentrations in streams and fish in the Blackfoot River basin in southeastern Idaho (Rich, 1999; MW, 1999, 2000). A preliminary hazard assessment of selenium revealed that selenium concentrations in fish exceeded the threshold for reproductive effects at one location and approached it at two other locations (Lemly, 1999a). Field studies at 18 sites including tributaries in the Blackfoot, Salt, and Bear river watersheds revealed moderate to high hazards at several sites (Hamilton et al., 2002b; Hamilton and Buhl, 2003a,b). Others have also expressed concern about the potential for adverse selenium impacts on fish and wildlife populations in the Blackfoot River basin because of elevated selenium concentrations in water, sediment, vegetation, fish, and bird eggs (Piper et al., 2000).

4.1. Food chain

The results of many of the selenium contaminant investigations discussed above have added substantially to the selenium literature in recent years. One major conclusion has been that selenium expresses its toxicity in animals primarily through the food chain (e.g. Maier and Knight, 1994; Lemly, 1996a). Some investigators have gone so far as to exclude water borne studies from consideration in discussing potential selenium toxicity thresholds in fish (DeForest et al., 1999).

A peer consultation workshop sponsored by the USEPA in 1998 advocated the use of a tissue-based selenium criterion because of the relation of between whole-body residues and adverse effects in fish (USEPA, 1998). Two other proposed criterion approaches, a sediment-based criterion and a water-based criterion, were not favored due to inadequacies. The water-based criterion was thought to be a poor choice because of temporal changes in concentrations, speciation, and rates of transfer between water, sediment and organisms, and a sediment-based criterion was a poor choice because of the spatial heterogeneity of deposition, variable water retention and volatilization rates, heterogeneity of benthic communities, and variable feeding habits of higher trophic organisms (reviewed in Hamilton, 2002). This workshop was a prelude to USEPA's revision of the selenium criteria for freshwater aquatic life. A draft selenium criterion using a tissue-based approach was issued to peer reviewers in the summer of 2002, but has not yet been published in the Federal Register for public comment due to concerns about protecting fish and wildlife resources in California. Nevertheless, the basis of the newly proposed USEPA tissue-based criterion is information from dietary selenium exposures. The tissue criterion approach accounts for selenium's biogeochemical pathways because it integrates the route (water and diet), duration and magnitude of exposure, chemical

form, metabolic transformations, and modifying biotic and abiotic factors. A rationale for a tissue-based criterion has been published (Hamilton, 2002).

4.2. Forms of selenium

Sandholm et al. (1973) were the first to demonstrate that selenium accumulation in fish was greater from dietary sources such as phytoplankton or zooplankton than from water. They also reported that there was little difference in fish accumulation of selenite or selenomethionine in the food chain. However, later studies by Bryson et al. (1985) and Woock et al. (1987) reported that diets incorporating selenite were not as toxic to fish as diets incorporating selenomethionine.

In general, dietary studies with selenomethionine have reported that toxic responses in fish were similar to those in fish fed diets containing naturally incorporated selenium compounds. For example, Bryson et al. (1985) reported that selenocystine incorporated into a fish food diet produced adverse effects in bluegill comparable to selenium-laden zooplankton. Hamilton et al. (1990) demonstrated that a selenomethionine diet caused similar adverse effects in chinook salmon (*Oncorhynchus tshawytscha*) as a diet incorporating selenium-laden western mosquitofish (*Gambusia affinis*) collected from the San Luis Drain, which in the early 1980s emptied into Kesterson Reservoir, CA. Likewise, Heinz (1996) reported that selenomethionine diets fed to adult mallards (*Anas platyrhynchos*) resulted in toxic concentrations in eggs that decreased hatching success and increased teratogenicity at concentrations nearly identical to those associated with similar adverse effects in field studies.

The source of the naturally incorporated selenium is an important consideration in dietary selenium studies. Bell and Cowey (1989) reported that the digestibility and availability of selenium to Atlantic salmon (*Salmo salar*) was the least for fish meal (source not given) and followed the order from greatest availability to least: selenomethionine > selenite > selenocystine > fish meal. The comparability of selenium-laden fish meal diet and a selenomethionine-fortified diet in three studies with chinook salmon (Hamilton et al., 1990) compared to differential digestibility reported in Atlantic salmon (Bell and Cowey, 1989) may reflect species differences or fish meal differences. In the study by Hamilton et al. (1990) the authors noted in both their freshwater and brackish water studies with chinook salmon that fish growth was significantly reduced at lower concentrations and in shorter exposure periods in fish fed the diet made with the selenium-laden western mosquitofish fish meal compared to the selenomethionine diet, which contained a comparable amount of clean fish meal from western mosquitofish. They suggested that the slightly greater toxic effect in fish fed the selenium-laden fish meal diet could have been caused by three factors: (1) additional toxic elements accumulated in the western mosquitofish inhabiting the San Luis Drain such as boron, chromium and strontium; (2) other forms of organoselenium, such as selenocystine present in the western mosquitofish; or (3) differential uptake, distribution or elimination of the protein-bound organoselenium in the fish fed the western mosquitofish fish meal diet compared to fish fed the free-amino acid selenomethionine diet that contained a comparable amount of clean fish meal. Nevertheless, the overall effects of the two diets were similar.

Sources of selenium in dietary studies with birds are also an important consideration. Hoffman et al. (1996) and Heinz et al. (1996) reported that the order from most toxic to less for selenium forms was: selenomethionine (DL form), selenomethionine (L form found in nature), selenized wheat, selenized yeast. Heinz (1996) reviewed the selenium literature for birds and noted that higher dietary concentrations of selenite than selenomethionine had to be fed to mallards to harm reproduction, but lower selenium concentrations in eggs from selenite exposures compared to selenomethionine exposures were associated with harm.

Due to the similarity between selenomethionine and naturally selenium-laden food organisms, selenomethionine-fortified diets have been used in several studies to determine toxic effects to fish (Table 1) and birds (Table 2).

The waterborne toxicity of selenate is less than that of selenite in aquatic invertebrates (Brasher

Table 2
Selenium concentrations in birds exposed to selenium in the diet and adverse effects

Species, age	Selenium exposure conc. (diet: μg/g, water: μg/l)	Selenium form	Exposure period (days)	Liver selenium (μg/g, wet wt.)	Effects	Reference
Mallard						
Adult	10	SEM[a]	~60	4.7♀ 8.6♂ 4.6 egg	Embryo deformity, duckling production, teratogenic	Heinz et al., 1987 Hoffman and Heinz, 1988
Duckling	25	Selenite	~60	2.6♀ 5.0♂ 1.3 egg	Adult weight, egg laying, embryo deformity, duckling production, duckling weight at hatch	
	10	SEM[a]	~21	–	Duckling survival, oxidative stress	
	25	Selenite	~21	–	Duckling survival, weight at 21 day	
Mallard						
Duckling	20	SEM[a]	42	26	Body weight, food consumption, oxidative stress	Heinz et al., 1988 Hoffman et al., 1989
	20	Selenite	42	3.2	Body weight, food consumption, oxidative stress	
Mallard,						
Adult	8	SEM[a]	~50	9.1 11 egg	Duckling production, duckling survival, duckling growth, teratogenic	Heinz et al., 1989
Duckling	16	SEC[b]	~50	6.9 0.6 egg	No effect	
Mallard,						
Duckling	15	SEM[a], 22% Protein	28	11	No effects	Hoffman et al., 1991
	60	SEM[a], 22% Protein		56	Mortality, growth, histopathology	
	15	SEM[a], 7% Protein		22	Growth	
	60	SEM[a], 7% Protein		–	Mortality	
Mallard,						
Duckling	15	SEM[a], 22% Protein	28	15	No effects	Hoffman et al., 1992a
	60	SEM[a], 22% Protein		72	Mortality, growth, histopathology	
	15	SEM[a], 7% Protein		25	Growth	

Table 2 *(Continued)*

Species, age	Selenium exposure conc. (diet: μg/g, water: μg/l)	Selenium form	Exposure period (days)	Liver selenium (μg/g, wet wt.)	Effects	Reference
	60	SEM[a], 7% Protein		–	Mortality	
Mallard,						
Duckling	15	SEM[a], 22% Protein	28	12	Histopathology, oxidative stress	Hoffman et al., 1992b
	60	SEM[a], 22% protein		49	Growth, histopathology	
	15	SEM + Me[c], 22% protein		9	No effect	
	60	SEM + Me[c], 22% protein		40	Growth, histo, oxidative stress	
	15	SEM[a], 11% protein		13	Oxidative stress	
	60	SEM[a], 11% protein		–	Mortality	
	15	SEM + Me[d], 11% protein		13	Oxidative stress	
	60	SEM + Me[d], 11% protein		–	Mortality	
	15	SEM[a], 44% protein		16	Growth	
	60	SEM[a], 44% protein		55	Mortality, growth, histopathology	
Mallard						
Adult	10	SEM[a]	~45	31♀ 34♂ 37 egg	Hatching success, teratogenic, duckling production	Stanley et al., 1994
Duckling	10	SEM[a]	14	20	Growth, mortality	
Mallard,						
Adult	3.5	SEM[a]	~50	3.7 3.5 egg	No effects	Stanley et al., 1996
	7	SEM[a]	~50	6.2 7.1 egg	Weight change, hatching success	
Duckling	3.5	SEM[a]	14	2.8	No effects	
	7	SEM[a]	14	5.0	Weight	

Table 2 *(Continued)*

Species, age	Selenium exposure conc. (diet: μg/g, water: μg/l)	Selenium form	Exposure period (days)	Liver selenium (μg/g, wet wt.)	Effects	Reference
Mallard,						
duckling	30	SEM(DL)[e]	14	20	Mortality, growth, oxidative stress	Hoffman et al., 1996
	30	SEM(L)[f]		19	Mortality, growth, oxidative stress	Heinz et al., 1996
	15	Se-wheat[g]		11	No effects	
	30	Se-yeast[h]		9.9	No effects	
	30	SEM(DL)[i]		27	Growth	
	15	SEM(L)[j]		25	Growth	
	30	Se-yeast[k]		13	Growth	
Mallard,						
Adult	8.8	SEM[a]	~30	6.0 7.6 egg	Hatchlings produced, 7-d duckling weight, duckling deformities	Heinz and Hoffman, 1998
Mallard,						
adult male	8.8	SEM[a]	70	11.5	Oxidative stress	Hoffman and Heinz, 1998
Mallard,						
adult male	40	SEM[a]	112	87	Behavior, organ weight, molt	Albers et al., 1996
Japanese quail,						
adult female	6	Selenite	84–112	3.5[l]	Hatching success, chick deformities	El-Begearmi et al., 1977
	12		84–112	4.4[l]	Adult mortality; egg production, fertility, hatching success, chick deformities	

[a] SEM: selenomethionine (DL form) in commercial duck food.
[b] SEC: selenocystine (DL form) in commercial duck food.
[c] SEM+Me: selenomethionine (DL form) with added 0.42% methionine in commercial duck food.
[d] SEM+Me: selenomethionine (DL form) with added 0.21% methionine in commercial duck food.
[e] SEM(DL): selenomethionine (DL form) in wheat-based duck food.
[f] SEM(L): selenomethionine (L form) in wheat-based duck food.
[g] Se-wheat: wheat containing 20 μg/g selenium in wheat-based duck food.
[h] Se-yeast: yeast containing 1000 μg/g in wheat-based duck food.
[i] SEM(DL): selenomethionine (DL form) in commercial duck food.
[j] SEM(L): selenomethionine (L form) in commercial duck food.
[k] Se-yeast: yeast containing 1000 μg/g in commercial duck food.
[l] Residue measured in liver after 4 week exposure.

and Ogle, 1993; Maier et al., 1993) and fish (Niimi and LaHam, 1976; Hamilton and Buhl, 1990). More importantly, selenite is taken-up faster and in greater amounts than selenate by aquatic plants (Maier et al., 1987; Vandermeulen and Foda, 1988; Riedel et al., 1991). Thus, selenite-dominated water bodies are more proficient at bioaccumulating selenium into the food chain than those that are selenate-dominated (Skorupa, 1998). Selenite-dominated aquatic ecosystems have been described as 'supercharged' with selenium compared to selenate-dominated systems (Skorupa, 1998).

4.3. Selenium interactions with other elements

Selenium interacts with several trace elements in fish, birds, and mammals (Diplock, 1976; Whanger, 1981; Marier and Jaworski, 1983; Sorensen, 1991). These interactions can be additive, antagonistic, or synergistic, and in some cases the interaction was reversed, i.e. antagonism changed to synergism. In general, selenium toxicity was alleviated by antimony, arsenic, bismuth, cadmium, copper, germanium, mercury, silver and tungsten (Diplock, 1976; Levander, 1977; Whanger, 1981; Marier and Jaworski, 1983), whereas chromium, cobalt, fluorine, molybdenum, nickel, tellurium, uranium, vanadium and zinc apparently have no effect on selenium toxicity (Hill, 1975; Ewan, 1978). Ohlendorf et al. (1993) reported on the uptake and potential interaction in wildlife of selenium with arsenic, boron, and molybdenum from food chain organisms in areas in central California impacted by seleniferous agricultural irrigation drain water.

Arsenic compounds have been shown to protect against the toxicity of a variety of forms of selenium including selenite, selenocystine, and selenomethionine (Levander, 1977). The protective effect of arsenic has been observed in rats, dogs, swine, cattle, and birds (Levander, 1977). In general, arsenic exposure in water or diet protected against dietary selenium toxicity (Moxon, 1938; Dubois et al., 1940; Klug et al., 1949, 1950; Levander and Argrett, 1969; Thapar et al., 1969; Howell and Hill, 1978), but combined arsenic and selenium waterborne exposure did not (Cabe et al., 1979; Frost, 1981). Dubois et al. (1940) and Klug et al. (1949) reported that the toxicity of selenite, selenomethionine, selenocystine, and seleniferous grain was reduced in rats by exposure to arsenic as either arsenite or arsenate, but not as arsenic sulfides. Klug et al. (1950) reported that arsenic protected rats against selenium-induced mortality, reduced growth, and reduced feeding, even though selenium residues were increased in liver (28%), kidney (141%), and muscle (52%) compared to exposure to only selenium in the diet (no arsenic exposure). Similar interactions between selenium and arsenic have been reported in dietary selenium studies with mallards (Hoffman et al., 1992a; Stanley et al., 1994) and razorback sucker (Hamilton et al., 2001b).

A few studies have reported interactions between selenium and copper in fish, and have observed altered residues dynamics, but not biological effects. Lorentzen et al. (1998), Berntssen et al. (1999, 2000) both reported that elevated dietary copper reduced the concentrations of selenium in liver of Atlantic salmon. Lorentzen et al. (1998) suggested that reduced selenium concentrations were due to the formation of insoluble copper–selenium complexes in the intestinal lumen, reducing selenium bioavailability or the excretion of copper–selenium complexes from the liver through the bile. Berntssen et al. (2000) reported that dietary copper exposure significantly reduced selenium concentrations in intestine and liver, which in turn reduced glutathione concentrations (selenium is a component of glutathione).

A couple of studies have examined the interactive effects of selenium and boron on mallard ducklings (Hoffman et al., 1991; Stanley et al., 1996). Although boron and selenium individually affected mallard reproduction and duckling growth, there was minimal interaction.

Perhaps one of the most published interactions between inorganic elements is that between mercury and selenium. Pelletier (1985) reviewed the literature for aquatic organisms and concluded that many authors reported simultaneous bioaccumulation of mercury and selenium, but there was no evidence of natural joint bioaccumulation of mercury and selenium in fishes, crustaceans or mollusks.

A series of experiments by Rudd, Turner, and others (Rudd et al., 1980; Turner and Rudd, 1983; Turner and Swick, 1983) investigated the ability of selenium to ameliorate the toxic effects on fish inhabiting a mercury-contaminated lake. They conducted enclosure experiments in the lake and reported that selenium additions reduced mercury accumulation in fish. Selenium interfered with mercury being mobilized through the food web rather than mercury accumulating directly from water. Klaverkamp et al. (1983) reported that exposure of northern pike (*Esox lucius*) to waterborne selenium at 1 μg/l reduced mercury accumulation in carcass, but exposure to 100 μg/l selenium increased mercury accumulation in carcass.

Another series of experiments in mercury-contaminated lakes in Sweden also tested the ameliorating effects of selenium (Paulsson and Lundbergh, 1989, 1991, 1994; Lindqvist et al., 1991). Similar to the studies by Rudd, Turner, and others, these Swedish studies also confirmed that selenium readily reduced mercury accumulation in fish, but selenium bioaccumulated in fish via the food chain if waterborne selenium concentrations were greater than 3–5 μg/l.

A significant delay in mortality, based on the predicted time-to-death, occurred in three studies with razorback sucker larvae conducted in different years and was thought due to an interaction of selenium and other elements (Hamilton et al., 1996, 2001a,b). In an experiment with 5-day-old larvae fed food organisms collected from various sites at Ouray National Wildlife Refuge on the Green River, UT, conducted in 1994, razorback sucker larvae held in water from Sheppard Bottom pond 1 (<1 μg/l selenium in water) and fed food organisms from either high-selenium North Roadside Pond (44 μg/g selenium) or South Roadside Pond (96 μg/g selenium) lived slightly longer (estimated median time to death: 14 days for North Roadside Pond and 15 days for South Roadside Pond) than larvae fed food organisms from low-selenium Sheppard Bottom pond 1 (3.5 μg/g selenium and 10 days) (Hamilton et al., 1996). In an experiment with 5-day old razorback sucker larvae conducted in 1996, larvae fed zooplankton from North Pond containing 39 μg/g selenium had estimated median time to death of 5.6 days in the reference water treatment (<1 μg/l), which was significantly longer than the 4.4 days in the site water treatment (10.7 μg/l) (Hamilton et al., 2001a). In an experiment with 5-day old razorback sucker larvae conducted in 1997, larvae fed zooplankton from Horsethief east wetland containing 4.6 to 8.1 μg/g selenium had estimated median time to death of 8 days in Horsethief water (1.6 μg/l selenium in water), 10 days in Adobe Creek water (3.4 μg/l selenium in water), and 14 days in North Pond water (13.3 μg/l selenium in water) (Hamilton et al., 2001b). Likewise, in the same study, there were other longer estimated median time to death in three food treatments and four different water treatments (Hamilton et al., 2001b). These examples of delayed mortality suggest an interaction between selenium in food and other elements in water.

Studies with mallard adults demonstrated that selenium and mercury were antagonistic (Heinz and Hoffman, 1998; Hoffman and Heinz, 1998). In contrast, combined selenium and mercury exposure was worse than individual exposures for mallard ducklings, and lowered duckling productivity through reduced hatching success, reduced survival, and increased teratogenic deformities (Heinz and Hoffman 1998; Hoffman and Heinz 1998). Studies with Japanese quail (*Coturnix coturnix*) exposed to selenium and methyl mercury in the diet have also demonstrated an antagonistic interaction (Stoewsand et al., 1974; El-Begearmi et al., 1977).

Several studies have been published elucidating the influence of nutritional factors on selenium toxicity in mallards. For example, in mallard ducklings reduced dietary protein increased selenium toxicity and increased methionine reduced selenium-induced mortality (Hoffman et al., 1991, 1992a,b).

4.4. Effects on biota

There has been a lack of consistency of adverse effects from selenium exposure on either growth or survival of fish, especially early life stages. Some fish studies with selenium exposure in the water, diet, or both have reported inconsistent

results: (1) reduced growth occurred in the same treatments (exposure concentration and duration) where reductions in survival occur (Hilton et al., 1980; Klauda, 1986); (2) reduced survival occurred before reduced growth (Hunn et al., 1987; Woock et al., 1987; Hamilton et al., 1990 [San Luis Drain diet]; Crane et al., 1992; Hermanutz et al., 1992; Cleveland et al., 1993); (3) reduced growth occurred before reduced survival (Hilton and Hodson, 1983; Ogle and Knight, 1989; Hamilton et al., 1990 [selenomethionine diet]); or (4) no effects on growth or survival, but other pathological or reproductive effects occurred (Hodson et al., 1980; Coyle et al., 1993). The inconsistency between these studies was probably due to differences in species, age, exposure route and duration, selenium form and other factors.

Teratogenesis is a well-documented biomarker of selenium toxicity in wild birds and fish at the embryo-larval stage (Ohlendorf et al., 1986a; Hoffman et al., 1988; Hoffman and Heinz, 1988; Lemly, 1993b, 1997a,c). Fish deformities include lordosis (concave curvature of lumbar and caudal regions of spine), kyphosis (convex curvature of thoracic region of the spine), scoliosis (lateral curvature of the spine), and head, mouth, gill cover, and fin deformities, in addition to edema, and brain, heart and eye problems. Selenium-induced teratogenic deformities in fish larvae have reported in laboratory studies (Goettl and Davies, 1977; Bryson et al., 1984; Klauda, 1986; Woock et al., 1987; Pyron and Beitinger, 1989), experimental stream studies (Schultz and Hermanutz, 1990; Hermanutz et al., 1992; Hermanutz, 1992), artificial crossing experiments (Gillespie and Baumann, 1986), and field investigations (Lemly, 1993b, 1997a,c; Saiki and Ogle, 1995; Hamilton et al., 2001a,b). Contaminated ecosystems may require long time periods for recovery from selenium contamination because 10 years after selenium inputs to Belews Lake, NC, were stopped, elevated incidences of deformed fry of four fish species were reported (Lemly, 1997a). Fish larvae exhibiting deformities would not be expected to survive in natural systems, except in predator-free situations (Hermanutz, 1992; Lemly, 1993b). Deformities have even been documented in selenium-tolerant western mosquitofish from the San Luis Drain, CA, where waterborne concentrations were 340–390 μg/l, and fish had over 100 μg/g selenium (Saiki and Ogle, 1995). Lemly (1993b) documented selenium-induced deformities in 19 species of fish from Belews Lake, NC, and reported that selenium residues were similar in normal-appearing fish and abnormal fish. Lemly (1997c) developed a teratogenic index for selenium-induced deformities in larval fish.

Elevated selenium concentrations in liver, kidney, ovaries, and testes have been linked with adverse pathological changes in those tissues along with lowered hematocrit and altered condition factor (Sorensen and Bauer, 1983; Sorensen et al., 1984; Sorensen, 1986, 1988, 1991).

Perhaps the most prominent and well documented effects of selenium in the food chain has been the elimination of fish species from aquatic ecosystems such as in Belews Lake, NC (Cumbie and Van Horn, 1978; Lemly, 1985), Martin Lake, TX (Garrett and Inman, 1984; Sorensen, 1988), Kesterson Reservoir, CA (Harris, 1986; Vencil, 1986), and the lack of reproduction as documented at Sweitzer Lake, CO (Barnhart, 1957; Birkner, 1978).

Measures of oxidative stress in birds have been extensively used in various bird populations to assess the potential impacts of selenium (reviewed in Hoffman, 2002). One or more of the oxidative measures from selenium exposure have been associated with teratogenesis (4.6 μg/g selenium wet weight in eggs), reduced growth in ducklings (15 μg/g in liver), diminished immune function (5 μg/g in liver), or histopathological lesions (29 μg/g in liver) (Hoffman, 2002). Manifestations of selenium-related effects on oxidative stress were apparent in field studies in seven species of aquatic birds (American avocet *Recurvirostra americana* and black-necked stilt in Tulare Basin, CA; American coot in Kesterson Reservoir, CA; surf scoter and ruddy duck *Oxyura jamaicensis* in San Francisco Bay, CA; emperor geese *Philacte canagica*in Western Alaska; willet *Catoptrophorus semipalmatus* in San Diego, CA; Hoffman, 2002).

5. Depuration

Several investigators have measured the depuration of selenium from fish. Most depuration

estimates for small fish range from 20 to 30 days for the half-life of selenium (Gissel Nielsen and Gissel-Nielsen, 1978; Sato et al., 1980; Hilton et al., 1982; Bennett et al., 1986; Besser et al., 1993), whereas Adams (1976) estimated a half-life of 63 days in whole body of adult fathead minnow and muscle of rainbow trout. Bertram and Brooks (1986) reported a half-life of approximately 49 days for adult fathead minnows exposed to selenium in the diet. In sub-adult bluegill and largemouth bass (*Micropterus salmoides*) with elevated selenium concentrations in tissues, Lemly (1982) noted no significant decrease in selenium concentrations in muscle, liver, kidney and spleen after 30 days depuration, which suggested a half-life much greater than 30 days. Similarly, Bryson et al. (1984) reported a half-life of selenium residues in adult bluegill of approximately 60 days. In a field study with large razorback sucker (approx. 1000 g) conducted in the upper Colorado River, the half-life of selenium depuration was greater than 100 days (Hamilton et al., 2001b). In contrast, changes in selenium residues does not seem to be occurring in large, wild endangered fish such as Colorado pikeminnow (approx. 1200 to 4000 g) in the upper Colorado River because fish recaptured over a 2 or 3-year period conserved selenium concentrations in muscle plugs from year to year (Osmundson et al., 2000).

An interesting example of depuration was given in Birkner (1978) who conducted a 90-day study with juvenile fathead minnow that initially had a whole-body selenium concentration of 13.9 μg/g. After 90 days of exposure, fish fed zooplankton with selenium concentrations of 1.2 μg/g had whole-body residues of 5.0–5.7 μg/g, those fed zooplankton with 5.7 μg/g had a whole-body residue of 5.2–7.0 μg/g, and those fed zooplankton with 11.8 μg/g had a whole-body residue of 10.3–11.0 μg/g. In a sense, fish achieved a new homeostasis through reduced intake and depuration of selenium from their initial whole-body residue to close to the concentration in their food.

Exposure to different forms of selenium seem to result in different total selenium half-lives. Kleinow and Brooks (1986) reported a 19-day half-life for selenate and selenite, and a 27-day half-life for selenomethionine in whole body of adult fathead minnow. They also reported that the half-lives were longer in muscle tissue: 33 days for selenate, 41 days for selenite, and 42 days for selenomethionine. The longer half-life for selenomethionine was probably due to its incorporation into protein and tissue, which would require more metabolic work to eliminate.

Overall, depuration of selenium from tissues depends on several factors including cleanliness of the food and water in the depurating environment, age, size, metabolic activity, season for poikilotherms, initial selenium load of various tissues, and other factors.

6. Species sensitivity

There seems to be clear evidence for differences in species sensitivity to selenium in both fish and birds. A good example is the elimination of some but not all species from aquatic communities impacted by selenium. Perhaps the best example of species elimination due to selenium contamination occurred at Belews Lake, NC, where 16 fish species disappeared (white sucker *Catostomus commersoni*, redbreast sunfish *Lepomis auritus*, pumpkinseed *Lepomis gibbosus*, warmouth *Lepomis gulosus*, bluegill, redear sunfish, largemouth bass, white crappie *Pomoxis annularis*, black crappie *Pomoxis nigromaculatus*, blueback herring *Alosa aestivalis*, threadfin shad *Dorosoma petenense*, golden shiner *Notemigonus chrysoleucas*, flat bullhead *Ictalurus platycephalus*, channel catfish *Ictalurus punctatus*, white perch *Morone americana*, yellow perch), three species persisted (common carp *Cyprinus carpio*, black bullhead *Ictalurus melas*, mosquitofish *G. affinis*), two species were introduced and reproduced (fathead minnow, red shiner *Notropis lutrensis*), and one species recolonized the lake (green sunfish) (Lemly, 1985).

Fish kills were observed at Martin Lake, TX, due to selenium contamination, and a 72% biomass decrease, excluding common carp, occurred as follows: planktivores changed from the largest to the smallest group, carnivores were initially reduced by half, and omnivores more than doubled (Garrett and Inman, 1984). Common carp, which persisted in Belews Lake, NC, had substantial

increases in biomass in Martin Lake. Specific biomass decreases were observed for gizzard shad (*Dorosoma cepedianum*) and threadfin shad, and reduced reproductive success was noted for largemouth bass. Assessments of redear sunfish conducted 8 years after the selenium contamination of Martin Lake reported seriously impaired reproductive status (Sorensen, 1988).

Following the introduction of selenium-laden irrigation drain water into Kesterson Reservoir, CA, populations of largemouth bass, striped bass, catfish species, and common carp disappeared and the only fish that persisted was the western mosquitofish (Vencil, 1986; NRC, 1989). Nevertheless, Saiki and Ogle (1995) reported that reproduction was impaired in western mosquitofish collected from the heavily selenium-contaminated San Luis Drain compared to a population from a reference site.

Sweitzer Lake located in the highly seleniferous Uncomphagre Valley and adjacent to the Uncomphagre River in Western Colorado was constructed in 1954 and stocked with eight game species including bluegill, channel catfish, minnows sp., white crappie, yellow walleye (*Stizostedion vitreum*), largemouth bass, black bullhead, and rainbow trout (Barnhart, 1957). Heavy mortalities were noted within a year of stocking and the only stocked fish to persist were black bullhead and channel catfish (Barnhart, 1957). Barnhart (1957) also noted spawning of fathead minnow, red shiner, central plains killifish (*Fundulus zebrinus*) and large schools of minnow fry. These fish are highly reproductive and could reproduce two to three times a year.

The pattern that seems to emerge from these fish studies is that a few tolerant species persist in selenium contaminant situations and sensitive species disappear due to direct mortality or reproductive failure. Aquatic ecosystems under stress tend to shrift from larger, longer-lived, benthic species to small, shorter-lived, non-native species (Rapport et al., 1985). Rapport et al. (1985) used the Great Lakes as an illustration: the larger, long-lived, benthic species included sturgeon sp., lake whitefish (*Coregonus clupeaformis*), lake trout (*Salvelinus namaycush*), walleye, northern pike, and the small, shorter-lived, non-native species included alewife (*Alosa pseudoharengus*) and rainbow smelt (*Osmerus mordax*). This pattern of species shrifts due to ecosystems stressed by selenium contamination seems to have occurred in the four examples above, i.e. general shrift from large, long-lived, native species to small, short-lived, non-native species, except for species tolerant of high stress situations like common carp and black bullheads.

Differences in selenium sensitivity have been reported for closely related birds. Black-necked stilt embryos seem to be more sensitive than American avocet embryos, and killdeer (*Charadrius vociferus*) embryos are more sensitive than snowy plover embryos (*Charadrius alexandrinus*) (Skorupa et al., 1996; Skorupa, 1998). There seems in birds examined thus far to be a relationship between selenium sensitivity and salt tolerance among related species. For example, black-necked stilts are more sensitive to selenium than are American avocets which are more salt tolerant, and sea ducks seem to tolerate much higher selenium exposures without apparent ill effects than do freshwater ducks, which are among the most sensitive of bird species to selenium (written communication, Schwarzbach, USGS). However, no examples of species shrifts based on size or life spans were found for birds that mirrored those reported for fish.

Some investigators believe the selenium literature is ambiguous about effects on fish. For example, fathead minnows have been used in selenium exposures and reported to have reduced survival and growth or reproductive failure including deformities in larvae at low dietary or water borne selenium concentrations in field studies (Schultz and Hermanutz, 1990; Hermanutz, 1992), but not in laboratory studies (Brooks et al., 1984; Ogle and Knight, 1989). Yet, fathead minnows survived and reproduced in Belews Lake, NC, and Sweitzer Lake, CO, which suggests they can tolerate stresses from selenium exposure, perhaps due to their rapid and high reproductive rate.

Likewise, some concerned people have pointed out that if some fish such as green sunfish, which recolonized Belews Lake, persist and reproduce in selenium stressed ecosystems, then the closely related bluegill should persist also. Differences in

vulnerability of closely related species, either birds or fish, to selenium stress probably depends primarily on their feeding niche or some adaptive, physiological mechanisms that in part makes closely related species separate species.

Similarly, some people have expressed the hypothesis that some species have adapted genetically, i.e. evolved, to high selenium environments. For example, it has been expressed that native fish in the Colorado River basin may have evolved in a selenium-rich environment because of the presence of high selenium soils derived from Cretaceous Mancos Shale. To address the issue of background waterborne selenium concentrations in streams and water bodies in Mancos Shale areas in the Grand and Uncompahgre valleys with no irrigation activity (some areas had grazing activity that disturbed the soils), David Butler of the US Geological Survey searched the area extensively and located seven areas for sampling (Butler and Osmundson, 2000). At six undisturbed sites with high selenium in the soil, low selenium concentrations (<1–1 μg/l) were found in several rainfall-runoff events. In addition, rainfall events generally had flow rates of 1–2 cfs, which yielded little volume in the arid habitat. In only one area with low flows (0.16–0.24 cfs) at West Salt Creek near S Road in the Grand Valley, selenium concentrations in water were elevated (9–10 μg/l) due to the presence of salt crusts. Since selenium concentrations in water draining high selenium soils in undisturbed areas were relatively low, i.e. $\sim$1 μg/l, the hypothesis of aquatic environments with elevated selenium, i.e. selenium enriched, enhancing the possibility of selenium adaptation by native fish seems unlikely.

Kennedy et al. (2000) hypothesized that cutthroat trout (*Oncorhynchus clarki*) had developed a tolerance to selenium in the Elk River of British Columbia, which is dominated by Cretaceous sedimentary formations, but they gave no supporting information. Although development of selenium tolerance of cutthroat trout was suggested in the Blackfoot River basin in Idaho, which is subject to selenium contamination from phosphate mining activities, genetic testing revealed no evidence for adaptation (MW, 2000). Though the hypothesis of selenium tolerance seems a possibility, no evidence has been published to support such a proposal.

6.1. Sediment effects

Sediments are an important consideration in field studies of selenium contamination. Several dietary selenium studies have been conducted with food organisms associated with sediments collected from selenium-contaminated environments. Woock (1984) demonstrated in a cage study with golden shiners that fish in cages with access to bottom sediments accumulated more selenium than fish held in cages suspended approximately 1.5 m above the sediments. This study showed that effects in fish were linked to selenium exposure via sediment, benthic organisms, detritus or a combination of sediment compartments. A similar finding was reported by Barnhart (1957) who reported that 'numerous species of game fish' lived at least 4 months when held in a livebox, which limited access to sediment, but fish lived less than 2 months when released in selenium-contaminated Sweitzer Lake, CO. The highly toxic nature of benthic organisms from selenium-contaminated Belews Lake, NC, was shown by Finley (1985) in an experiment where bluegill died in 17 to 44 days after being fed selenium-laden *Hexagenia* nymphs containing 13.5 μg/g wet weight.

6.2. Seasonal effects

An important aspect influencing the toxic effects in fish resulting from dietary selenium exposure is season (Lemly, 1996b, 1997b). The toxicity to bluegill of combined low dietary (5.1 μg/g) and low waterborne (4.8 μg/l) selenium at low water temperature (4 °C) resulted in significantly increased mortality of fish (Lemly, 1993a). The combination of a stress-related elevated energy demand from selenium exposure and reductions in feeding due to cold temperature and short photoperiod led to a severe depletion of stored body lipid and an energetic drain that resulted in the death of about a third of the fish tested. Heinz and Fitzgerald (1993a,b) also suggested that stress from winter conditions might have increased the

harmful effects of dietary selenium in adult mallards.

7. Ecosystem recovery

The concept of depuration is appropriate in controlled experiments, but may be misleading in the natural environment because laboratory measurements were on fish physically placed in a clean environment for the sole purpose of determining how fast their tissues can remove a contaminant. In the natural environment, aquatic invertebrates, fish, and wildlife may not be able to move to a clean environment once an aquatic site is contaminated. An example of aquatic ecosystem loading of selenium and lack of depuration in invertebrates was given by Maier et al. (1998). They reported that application of seleniferous fertilizer (1% selenium by weight as sodium selenite) to a deer forage range in California resulted in a pulse of selenium entering the stream and briefly raising the water selenium concentrations from <1 μg/l prior to application to 10.9 μg/l at 3 h post-application. Selenium concentrations in aquatic invertebrates in the stream increased from 1.67 μg/g before application to 4.74 μg/g 3 h after the application and remained elevated after 2 (4.02 μg/g), 4 (4.99 μg/g), 6 (4.21 μg/g), 8 (4.30 μg/g), and 11 months (4.54 μg/g), even though water concentrations were <1 μg/l between 11 days and 11 months post application. Their study was the first to show that a short pulse event can quickly load an aquatic environment with selenium, and that selenium could be conserved in the ecosystem.

Another example of the bioaccumulative effects of selenium was given by Crane et al. (1992) who treated ponds with 2 μg/l selenium for 288 days. This low selenium treatment resulted in selenium concentrations of 12.6 μg/g in aquatic insects, 8.6 μg/g in molluscs, and 14.6 μg/g in crustaceans. They noted no major differences in benthic invertebrate communities during their study. This scenario of aquatic ecosystem loading of selenium was also demonstrated at Adobe Creek, a diked tertiary channel in the Grand Valley of Western Colorado, during a reproduction study with razorback sucker (Hamilton et al., 2001a,b), and has been documented in San Francisco Bay, CA (Luoma and Presser, 2000).

Once selenium is present in an aquatic ecosystem, it is efficiently recycled through a multitude of compartments, i.e. surface water, ground water, sediments, porewater, detritus, bacteria, benthic invertebrates, detritus feeders, floating plants, rooted plants, algae, zooplankton, macroinvertebrates, fish, birds, and mammals (reviewed by Maier et al., 1987; Presser and Ohlendorf, 1987; Davis et al., 1988; Maier et al., 1988; Ogle et al., 1988; Ohlendorf, 1989; Maier and Knight, 1994; Lemly, 1996a). Since the burial of Kesterson Reservoir and its selenium-contaminated sediments, winter rains have created ephemeral pools whose waters contained selenium concentrations up to 1600 μg/l (reviewed by Presser and Piper, 1998). Aquatic invertebrates in these pools contained geometric mean selenium concentrations of 8.5–12.5 μg/g, which showed that burial of selenium-contaminated soil might not stop the recycling of selenium nor reduce its availability to biota.

Sorensen and Bauer (1984) reported that 2 years after selenium inputs to Martin Lake, TX, were stopped, selenium concentrations in ovary of redear sunfish were 20–24 μg/g (reported as wet weight, converted to dry weight assuming 75% moisture), which is two times higher than the toxicity threshold of 10 μg/g in ovary or eggs (Lemly, 1996a). Sorensen (1988) reported that selenium tissue residues in fish from Martin Lake, TX, were only 25% lower after a 5-year period (1981–1986) following the reduction of selenium inputs to the lake between 1978 and 1981.

Likewise, Lemly (1997a) assessed selenium concentrations in five ecosystem components of Belews Lake, NC, 10 years after selenium inputs to the lake were stopped and found elevated selenium concentrations in sediment, benthic invertebrates, and fish that suggested a moderate hazard still existed. He also reported teratogenic deformities first observed in 1992 (Lemly, 1993b) were still present at elevated levels in 1996.

8. Selenium thresholds

There seems to be a general convergence of laboratory and field results leading to a consensus

Table 3
Selenium thresholds (dry weight)

Medium	No effect[a]	Level of concern[b]	Toxicity threshold[c]	Reference
Water (μg/l)	<2	–	>2.7	Maier and Knight, 1994
	–	–	2	Lemly, 1996a
	<2	2–5	>5	Henderson et al., 1995
	<1	1–2	>2	Stephens et al., 1997
	<1	1–2	>2	USDOI, 1998
	<2	2–5	>5	URS, 2000
Sediment (μg/g)	<2	2–4	>4	Henderson et al., 1995
	<2	2–4	>4	Stephens et al., 1997
	<1	1–4	>4	USDOI, 1998
	<2	2–4	>4	URS, 2000
Diet (μg/g)	<3	–	>4	Maier and Knight, 1994
	–	–	3	Lemly, 1996a
	<3	3–7	>7	Henderson et al., 1995
	<2	2–3	>3	Stephens et al., 1997
	<2	2–3	>3	USDOI, 1998
	–	–	10 warmwater 11 coldwater	DeForest et al., 1999
	<3	3–7	>7	URS, 2000
	–	–	3	Hamilton, 2003
Waterbird eggs (μg/g)	<3	3–8	>8	Henderson et al., 1995
	–	–	10	Heinz, 1996
	<3	3–8	>8	Stephens et al., 1997
	<3	3–6	>6	USDOI, 1998
	–	–	>6	Skorupa, 1998
	–	–	16[d]	Fairbrother et al., 1999
	<6	6–10	>10	URS, 2000
	–	10[c]	>10	Spallholz and Hoffman, 2002
liver	–	–	33[e]	Heinz, 1996
liver[f]	–	–	10[e]	Heinz, 1996
Fish, whole-body (μg/g)	<3	–	>4.5	Maier and Knight, 1994
	–	–	4	Lemly, 1996a
	<4	4–12	>12	Henderson et al., 1995
	<2–3	4	>4	Stephens et al., 1997
	<2–3	2–4	>4	USDOI, 1998
	–	–	6 coldwater 9 warmwater	DeForest et al., 1999
	<4	4–9	>9 warmwater	URS, 2000
	–	–	>4	Hamilton, 2002

[a] Concentrations less than this value produce no discernible adverse effects on fish or wildlife and are typical of background concentrations in uncontaminated environments (USDOI, 1998).
[b] Concentrations in this range rarely produce discernible adverse effects on some fish or wildlife species (USDOI, 1998).
[c] Concentrations greater than this value seem to produce adverse effects on some fish or wildlife species (USDOI, 1998).
[d] EC10 value (effect concentration at 10% level).
[e] Dry weight concentration converted from wet weight assuming 70% moisture (Heinz et al., 1989).
[f] Laying females.

among government and academic researchers in most selenium thresholds for adverse effects in fish and birds (Table 3). For fish, the selenium dietary concentration associated with reduced growth or survival is generally close to 3 μg/g, and the whole-body selenium residue close to 4 μg/g (Table 1).

Heinz (1996) reviewed the selenium literature for birds and concluded that selenium concentrations in eggs were better predictors of adverse effects on reproduction than were concentrations in liver (Table 2). He recommended a threshold concentration in eggs of 10 μg/g (converted from wet weight using 70% moisture). Nevertheless, he also recommended a threshold concentration in liver of young or adult birds of 33 μg/g (converted), and 10 μg/g in liver of laying hens (converted).

Many of the references in Tables 1 and 2 were the foundation for deriving the thresholds summarized in Table 3. For water, most proposed thresholds are below the current national water quality criterion for the protection of aquatic life of 5 μg/l (USEPA, 1987). The basis for the lower concentrations is the bioaccumulation of low water borne selenium in the food web to dietary concentrations above proposed toxicity thresholds.

For sediment there seems to be a general consensus among government and academic researchers at 4 μg/g for the toxicity threshold. However, several publications have reported very elevated selenium concentrations in benthic invertebrates collected from water and sediments with low selenium concentrations. For example, Birkner (1978) collected samples at four sites in Colorado and Wyoming that contained selenium concentrations of 0.7 to 4.2 μg/l in water, 1.2 to 3.3 μg/g in sediments, and 4.4 to 28.4 μg/g in aquatic invertebrates. As another example, Zhang and Moore (1996) collected samples at Benton Lake, MT, that contained selenium concentrations of 0.9 to 1.6 μg/l in water, 0.4 to 1.4 μg/g in sediment, and 8.1 to 10.4 μg/g in chironomid larvae. In both of these examples, selenium concentrations were low in water and sediments, but substantially elevated in invertebrates above the dietary toxicity threshold of 3 μg/g. Consequently, a thorough evaluation in the future of the relation between selenium concentrations in sediments and benthic invertebrates may result in lower sediment selenium toxicity thresholds.

For diet, most proposed threshold values are close to 3–4 μg/g. One recent publication by DeForest et al. (1999) has proposed dietary selenium toxicity thresholds of 10 μg/g for warmwater fish and 11 μg/g for coldwater fish, which are three times higher than proposed by others. They also separated the whole-body residue thresholds for adverse effects for cold and warm water fish and proposed 6 μg/g and 9 μg/g, respectively. DeForest et al. (1999) based their proposed values on a limited dataset, whereas a review of a large selenium dataset seems to lack support for separating thresholds for cold and warm water fish or for elevated dietary threshold (Hamilton, 2003).

For waterbird eggs there seems to be a consensus among government researchers for a toxicity threshold at approximately 6–10 μg/g. Researchers such as Heinz, Hoffman and Skorupa each have over 20 years of research experience and numerous publications dealing with selenium toxicity to waterbirds, thus their proposed thresholds are founded on substantial expertise. The one proposed high threshold by Fairbrother et al. (1999) has been critiqued by Skorupa (1999) and responded to by Fairbrother et al. (2000). Some of the controversy in selenium thresholds is due to different thresholds. Skorupa (1999) estimated an EC03 (effect concentration at the 3% level) threshold of 6 μg/g for viability of stilt eggs, whereas Fairbrother et al. (1999) estimated an EC10 threshold of 16 μg/g and an EC20 of 21 μg/g for duckling production (a composite egg fertility, viability of egg fertility, and early posthatch duckling survival). A discussion of selecting different EC endpoints and the effect of dataset selection on statistical results is given in Skorupa (1999).

For whole-body fish most researchers have proposed a toxicity threshold of approximately 4 μg/g. An earlier value proposed by Henderson et al. (1995); 12 μg/g) who were with the US Fish and Wildlife Service has since been lowered to 4 μg/g in guidelines of the DOI (USDOI 1998). DeForest et al. (1999) used a limited dataset to derive the slightly higher thresholds. URS (2000) essen-

tially borrowed their proposed whole-body fish thresholds, with cautionary caveats, from DeForest et al. (1999).

The threshold concentrations discussed above should not be considered safe concentrations because no safety factor has been incorporated. For regulatory purposes, a safety factor is added to account for some level of uncertainty (USEPA, 1984; Pendergast et al., 1997). Such cautions have been noted by others (Heinz, 1996; Skorupa, 1998).

8.1. Controversy among selenium thresholds

The criteria for selenium in freshwater ecosystems and interpretation of the selenium literature has become a controversial topic in recent years as evidenced by debate papers in the Journal Human and Ecological Risk Assessment (Chapman, 1999; Lemly, 1999b; Hamilton, 1999; Ohlendorf, 1999; DeForest et al., 1999; Fairbrother et al., 1999), response papers (Skorupa, 1999; Fairbrother et al., 2000), and debates at national scientific meetings, i.e. 'Selenium in the Environment: A Ticking Time Bomb or No Big Deal?' (SETAC 1999). There seems to be a divergence between academia or government-backed papers proposing low selenium criteria, and non-governmental papers proposing high criteria (reviewed in Hamilton, 2003). Most of the academia/government papers were published in the late 1980s and early 1990s with follow-up papers including the latest information in the late 1990s, whereas the non-governmental papers were published in the late 1990s. The effort by non-governmental entities to present their viewpoints seems to have coincided with the effort by USEPA to reevaluate the current selenium criterion for the protection of aquatic life. Wetland use as a remediation technique to reduce selenium bioaccumulation in higher tropic levels also continues to be a matter of debate (Lemly, 1999b; Ohlendorf and Gala, 2000; Lemly and Ohlendorf, 2002).

Comparability of selenium effects between lotic (flowing) and lentic (static) aquatic ecosystems has also been a point of controversy. Some investigators believe that the results from lentic studies, which typically have high bioaccumulation rates, are not comparable or applicable to lotic studies, which typically have low bioaccumulation rates (Kennedy et al., 2000; Adams et al., 2000). Other investigators believe that the interconnectedness of lotic (streams, rivers) and lentic (backwaters, side channels, reservoirs) areas cannot be separated from each other when assessing selenium impacts on aquatic resources (Skorupa, 1998; Hamilton and Lemly, 1999; Lemly, 1999b; Hamilton and Palace, 2001).

Another controversial area of the selenium literature that is starting to grow is the suggestion of different thresholds for cold water vs. warm water fish (DeForest et al., 1999; URS, 2000; Brix et al., 2000). Most of the literature is for warm water fish studies in lentic ecosystems, whereas there are fewer studies with cold water fish typically found in lotic ecosystems. A commentary paper has addressed these concerns and concluded that there is little evidence for a foundation for differentiating selenium thresholds between warm water and cold water fish (Hamilton, 2003).

9. Research needs

There is an extensive database on waterborne and dietary selenium toxicity to a variety of fish and a limited set of bird species. A limited number of studies have been conducted to elucidate the interaction of selenium and other elements or nutritional factors in fish and wildlife. A few studies have reported that other elements and nutritional factors influence selenium toxicity, but more studies are needed to further our limited knowledge of interactions in this area.

The emphasis in aquatic toxicology over the past few decades has been on waterborne exposures. Consequently, national water quality criteria have been propagated based on a waterborne approach including selenium. In recent years, laboratory studies with selenium have shown that dietary exposures are the major route of exposure for fish and wildlife. Information from field studies investigating selenium contaminated sites have demonstrated that diet can be an important contributor to toxic effects, if not the dominant factor, yet national water quality criteria have not considered the effects from dietary exposures. This over-

sight has been somewhat addressed in the effort by the US Environmental Protection Agency to establish a tissue-based selenium criterion for the protection of aquatic life. Unfortunately, this effort has been put on hold due to differences of opinion on the appropriate selenium concentration for a national tissue-based criterion. Specifics on the implementation of a tissue-based criterion need to be developed before it can be practically applied in the real world of water quality regulation.

Fewer selenium studies have been conducted with mammals and varieties of birds compared with fish. There are many unique species of wild mammals with varied sensitivities to certain classes of contaminants, and virtually none have been used in selenium studies. Consequently, there is a need for selenium studies with mammals other than livestock and with more bird species. Effects upon marine birds and adult migratory birds receiving transitory exposures along their migration route are also needed due to selenium's possible impact to mass wasting and required fitness for sustained flight of migration. Another area needing investigation is the effects of selenium on amphibians and reptiles.

The controversies mentioned concerning selenium effects in lotic (flowing) ecosystems, wetlands, cold water vs. warm water fish sensitivity, and seemingly inconsistent responses of some fish species such as fathead minnow call out for additional research to investigate these information gaps. Although the selenium database may seem large compared other elements, there are substantial information gaps that have continued to fuel controversies.

Acknowledgments

The author thanks William Beckon and Steve Schwarzbach for review comments, and Karen Faerber for typing drafts of the manuscript.

References

Adams WJ. The toxicity and residue dynamics of selenium in fish and aquatic invertebrates. Unpublished dissertation, Michigan State University, East Lansing, 1976.

Adams WJ, Toll JE, Brix KV, Tear LM, DeForest DK. Site-specific approach for setting water quality criteria for selenium: differences between lotic and lentic systems. In: Proceedings of the 24th Annual British Columbia Reclamation Symposium, BC Technical and Research Committee on Reclamation. Planning for End Land Use in Mine Reclamation, 19–22 June, Williams Lake, BC, 2000, pp. 231–240.

Albers PH, Green DE, Sanderson CJ. Diagnostic criteria for selenium toxicosis in aquatic birds: dietary exposure, tissue concentrations, and macroscopic effects. J Wildl Dis 1996;32:468–485.

Barnhart RA. Chemical factors affecting the survival of game fish in a Western Colorado Reservoir. Unpublished thesis, Colorado State University, Fort Collins, 1957.

Bell JG, Cowey CB. Digestibility and bioavailability of dietary selenium from fishmeal, selenite, selenomethionine and selenocystine in Atlantic salmon (*Salmo salar*). Aquaculture 1989;81:61–68.

Bennett WN, Brooks AS, Boraas ME. Selenium uptake and transfer in an aquatic food chain and its effects on fathead minnow larvae. Arch Environ Contam Toxicol 1986;15:513–517.

Berntssen MHG, Lundebye AK, Maage A. Effects of elevated dietary copper concentrations on growth, feed utilisation and nutritional status of Atlantic salmon (*Salmo salar* L.) fry. Aquaculture 1999;174:167–181.

Berntssen MHG, Lundebye AK, Hamre K. Tissue lipid peroxidative responses in Atlantic salmon (*Salmo salar* L.) parr fed high levels of dietary copper and cadmium. Fish Physiol Biochem 2000;23:35–48.

Bertram PE, Brooks AS. Kinetics of accumulation of selenium from food and water by fathead minnows. Water Res 1986;20:877–884.

Besser JM, Huckins JN, Little EE, La Point TW. Distribution and bioaccumulation of selenium in aquatic microcosms. Environ Pollut 1989;62:1–12.

Besser JM, Canfield TJ, La Point TW. Bioaccumulation of organic and inorganic selenium in a laboratory food chain. Environ Toxicol Chem 1993;12:57–72.

BISF (Bay Institute of San Francisco). Selenium and Agricultural Drainage: implications for San Francisco Bay and the California Environment. Proceedings of the Second Selenium Symposium, The Bay Institute of San Francisco, Tiburon, CA, 1986.

Birkner JH. Selenium in aquatic organisms from seleniferous habitats. Unpublished dissertation, Colorado State University, Fort Collins, 1978.

Bowie GL, Sanders JG, Riedel GF, Gilmour CC, Breitburg DL, Cutter GA, Porcella DB. Assessing selenium cycling and accumulation in aquatic ecosystems. Water Air Soil Pollut 1996;90:93–104.

Brasher AM, Ogle RS. Comparative toxicity of selenite and selenate to the amphipod *Hyalella azteca*. Arch Environ Contam Toxicol 1993;24:182–186.

Brix KV, DeForest DK, Fairbrother A, Adams WJ. Critical review of tissue-based selenium toxicity thresholds for fish and birds. In: Proceedings of the 24th Annual British Columbia Reclamation Symposium, BC Technical and

Research Committee on Reclamation. Planning for End Land Use in Mine Reclamation, 19–22 June, Williams Lake, BC, 2000, pp. 220–230.

Brooks AS, Bertram PE, Szmania DC, Seale DB, Boraas ME, Kleinow KM, Boyum K, Jones-Witthuhn R, Bennett W. The effect of selenium on the reproductive potential of the fathead minnow. Final report to the Electric Power Research Institute, Palo Alto, CA, Research Project 1631-1, University of Wisconsin, Milwaukee, 1984.

Bryson WT, Garrett WR, Mallin MA, MacPherson KA, Partin WE, Woock SE. Roxboro steam electric plant environmental monitoring studies 1982. Hyco reservoir bioassay studies, vol. II. New Hill, NC: Carolina Power and Light, 1984.

Bryson WT, MacPherson KA, Mallin MA, Partin WE, Woock SE. Roxboro Steam Electric Plant Hyco Reservoir 1984 bioassay report. Carolina Power and Light Company, New Hill, NC, 1985.

Butler DL. Effects of piping irrigation laterals on selenium and salt loads, Montrose Arroyo basin, Western Colorado. US Geological Survey Water-Resources Investigations Report 01-4204, Denver, CO, 2001, p. 14.

Butler DL, Osmundson BC. Physical, chemical, and biological data for the Uncompahgre Project Area and the Grand Valley, west-central Colorado, 1993–1998. US Geological Survey Open-File Report 99-453, Denver, CO, 2000.

Cabe PA, Carmichael NG, Tilson HA. Effects of selenium, alone and in combination with silver or arsenic, in rats. Neurobehav Toxicol 1979;1:275–278.

Caribou County Sun. Toxicologist and vet say dead sheep likely died from selenium. Caribou County Sun, Soda Springs, ID, 11 November 1999.

CH2M Hill. Selenium bioaccumulation study at Chevron's Richmond refinery water enhancement wetland. Oakland, CA: CH2M Hill, 1995.

Chapman PM. Selenium – a potential time bomb or just another contaminant? Human Ecol Risk Assess 1999;5:1123–1138.

Cherry DS, Guthrie RK. Toxic metals in surface waters from coal ash. Water Res Bull 1977;13:1227–1236.

Clark DR Jr. Selenium accumulation in mammals exposed to contaminated California irrigation drainwater. Sci Total Environ 1987;66:147–168.

Clark DR Jr, Ogasawara PA, Smith GJ, Ohlendorf HM. Selenium accumulation by raccoons exposed to irrigation drainwater at Kesterson National Wildlife Refuge, California, 1986. Arch Environ Contam Toxicol 1989;18:787–794.

Cleveland L, Little EE, Buckler DR, Wiedmeyer RH. Toxicity and bioaccumulation of waterborne and dietary selenium in juvenile bluegill (*Lepomis macrochirus*). Aquat Toxicol 1993;27:265–280.

Coughlan DJ, Velte JS. Dietary toxicity of selenium-contaminated red shiners to striped bass. Trans Am Fish Soc 1989;118:400–408.

Coyle JJ, Buckler DR, Ingersoll CG, Fairchild JF, May TW. Effect of dietary selenium on the reproductive success of bluegills (*Lepomis macrochirus*). Environ Toxicol Chem 1993;12:551–565.

Crane M, Flower T, Holmes D, Watson S. The toxicity of selenium in experimental freshwater ponds. Arch Environ Contam Toxicol 1992;23:440–452.

Cumbie PM, Van Horn SL. Selenium accumulation associated with fish mortality and reproductive failure. Proceedings of Annual Conference of Southeastern Assoc Fish Wildlife Agencies 1978; 32 pp. 612–624.

Cutter GA. The estuarine behaviour of selenium in San Francisco Bay. Estuar Coastal Shelf Res 1989;28:13–34.

Dallinger R, Prosi F, Segner H, Back H. Contaminated food and uptake of heavy metals by fish: a review and a proposal for further research. Oecologia 1987;73:91–98.

Darnall NL, Waddell B, Boeke E. Remediation activities at Stewart Lake and selenium reductions in biota. In: Society of Environmental Toxicology and Chemistry 23rd Annual Meeting Abstract Book, November 16–20, 2002, Salt Lake City, UT, 2002, Abstract P329, p. 225.

Davis EA, Maier KJ, Knight AW. The biological consequences of selenium in aquatic ecosystems. Calif Agr 1988;42:18–20, 29.

DeForest DK, Brix KV, Adams WJ. Critical review of proposed residue-based selenium toxicity thresholds for freshwater fish. Human Ecol Risk Assess 1999;5:1187–1228.

Desborough G, DeWitt E, Jones J, Meier A, Meeker G. Preliminary mineralogical and chemical studies related to the potential mobility of selenium and associated elements in phosphoria formation strata, southeastern Idaho. US Geological Survey Open-File Report 99-129, 1999.

Diplock AT. Metabolic aspects of selenium action and toxicity. CRC Crit Rev Toxicol 1976;5:271–329.

Dubois KP, Moxon AL, Olson OE. Further studies on the effectiveness of arsenic in preventing selenium poisoning. J Nutr 1940;19:477–482.

Eisler R. Selenium. Handbook of chemical risk assessment: health hazards to humans, plants, and animals, vol. 3. Boca Raton, FL: Lewis Publishers, CRC Press, 2000. p. 1649–1705.

El-Begearmi MM, Sunde ML, Ganther HE. A mutual protective effect of mercury and selenium in Japanese quail. Poultry Sci 1977;56:313–322.

Engberg RA. Determination of and planning remediation for selenium contamination of water, bottom sediment and biota by drainwater from federal irrigation projects. Interdisciplinary approaches in hydrology and hydrogeology. American Institute of Hydrology 1992. p. 184–194.

Engberg RA. Remediation of irrigation-related contamination at Department of the Interior project areas in the Western United States. In: Dudley LM, Guitjens JC, editors. Agroecosystems and the environment. San Francisco: Pacific Division of the American Association for the Advancement of Science, 1998. p. 57–76.

Engberg RA. Selenium budgets for Lake Powell and the upper Colorado River basin. J Am Water Res Assoc 1999;35:771–786.

Engberg RA, Sylvester MA. Concentrations, distribution, and sources of selenium from irrigated lands in Western United States. J Irrig Drain Eng 1993;119:522–535.

Ewan RC. Toxicology and adverse effects of mineral imbalance with emphasis on selenium and other minerals. In: Oehme FW, editor. Toxicity of heavy metals in the environment, part 1. New York: Marcel Dekker, 1978. p. 445–489.

Fairbrother A, Brix KV, Toll JE, McKay S, Adams WJ. Egg selenium concentrations as predictors of avian toxicity. Human Ecol Risk Assess 1999;5:1229–1253.

Fairbrother A, Brix KV, DeForest DK, Adams WJ. Egg selenium thresholds for birds: a response to J. Skorupa's critique of Fairbrother et al., 1999. Human Ecol Risk Assess 2000;6:203–212.

Feltz HR, Engberg RA. Historical perspective of the US Department of Interior National Irrigation Water Quality Program. In: Marston RA, Hasfurther VR, editors. Proceedings of the Annual Summer Symposium of the American Water Resources Association. Effects of Human-Induced Changes on Hydrologic Systems. Jackson Hole, WY, 1994, pp. 1011–1020.

Finley KA. Observations of bluegills fed selenium-contaminated *Hexagenia* nymphs collected from Belews Lake, North Carolina. Bull Environ Contam Toxicol 1985;35:816–825.

Frost DV. Selenium and vitamin E as antidotes to heavy metal toxicities. In: Spallholz JE, Martin JL, Ganther HE, editors. Selenium in biology and medicine. Westport, CT: AVI Publishing Company, 1981. p. 490–497.

Furr AK, Parkinson TF, Youngs WD, Berg CO, Gutenmann WH, Pakkala IS, Lisk DJ. Elemental content of aquatic organisms inhabiting a pond contaminated with coal fly ash. NY Fish Game J 1979;26:154–161.

Garrett GP, Inman CR. Selenium-induced changes in fish populations of a heated reservoir. Proceedings of Annual Conference of Southeastern Association Fish Wildlife Agencies 1984;38:291–301.

Gillespie RB, Baumann PC. Effects of high tissue concentrations of selenium on reproduction by bluegills. Trans Am Fish Soc 1986;115:208–213.

Gissel Nielsen M, Gissel-Nielsen G. Sensitivity of trout to chronic and acute exposure to selenium. Agr Environ 1978;4:85–91.

Goettl JP Jr, Davies PH. Water pollution studies. Job Progress Report, Federal Aid Project F-33-R-12, Colorado Division of Wildlife, Fort Collins, 1977.

Goettl JP Jr, Davies PH. Water pollution studies. Job Progress Report, Federal Aid Project F-33-R-13, Colorado Division of Wildlife, Fort Collins, 1978.

Greenberg AJ, Kopec D. Decline of bay-delta fisheries and increased selenium loading: possible correlation? In: Proceedings of the Second Selenium Symposium. Selenium and Agricultural Drainage: Implications for San Francisco Bay and the California Environment. The Bay Institute of San Francisco, Tiburon, CA, 1986, pp. 69–81.

Hamilton SJ. Selenium effects on endangered fish in the Colorado River basin. In: Frankenberger Jr WT, Engberg RA, editors. Environmental chemistry of selenium. New York: Marcel Dekker, 1998. p. 297–313.

Hamilton SJ. Hypothesis of historical effects from selenium on endangered fish in the Colorado River Basin. Human Ecol Risk Assess 1999;5:1153–1180.

Hamilton SJ. Rationale for a tissue-based selenium criterion for aquatic life. Aquat Toxicol 2002;57:85–100.

Hamilton SJ. Commentary: review of residue-based selenium toxicity thresholds for freshwater fish. Ecotoxicol Environ Safe 2003;56:201–210.

Hamilton SJ, Buhl KJ. Acute toxicity of boron, molybdenum, and selenium to fry of chinook salmon and coho salmon. Arch Environ Contam Toxicol 1990;19:366–373.

Hamilton SJ, Buhl KJ. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: September 2000. Final report as part of the USGS Western US Phosphate Project. US Geological Survey, Yankton, SD, 2003a, p. 63.

Hamilton SJ, Buhl KJ. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: May 2001. Final report as part of the USGS Western US Phosphate Project. US Geological Survey, Yankton, SD, 2003b, p. 61.

Hamilton SJ, Lemly AD. Commentary: water-sediment controversy in setting environmental standards for selenium. Ecotoxicol Environ Safe 1999;44:227–235.

Hamilton SJ, Wiedmeyer RH. Concentrations of boron, molybdenum, and selenium in chinook salmon. Trans Am Fish Soc 1990;119:500–510.

Hamilton SJ, Palace VP. Commentary: assessment of selenium effects in lotic ecosystems. Ecotoxicol Environ Safe 2001;50:161–166.

Hamilton SJ, Palmisano AN, Wedemeyer GA, Yasutake WT. Impacts of selenium on early life stages and smoltification of fall chinook salmon. Trans N Am Wildl Nat Res Conf 1986;51:343–356.

Hamilton SJ, Buhl KJ, Faerber NL, Wiedmeyer RH, Bullard FA. Toxicity of organic selenium in the diet to chinook salmon. Environ Toxicol Chem 1990;9:347–358.

Hamilton SJ, Buhl KJ, Bullard FA, McDonald SF. Evaluation of toxicity to larval razorback sucker of selenium-laden food organisms from Ouray NWR on the Green River, Utah. National Biological Service, Yankton, SD, Final Report to the Recovery Implementation Program for the Endangered Fishes of the Colorado River Basin, Denver, 1996, p. 79.

Hamilton SJ, Buhl KJ, Bullard FA, Little EE. Chronic toxicity and hazard assessment of an inorganic mixture simulating irrigation drainwater to razorback sucker and bonytail. Environ Toxicol 2000a;15:48–64.

Hamilton SJ, Muth RT, Waddell B, May TW. Hazard assessment of selenium and other trace elements in wild larval razorback sucker from the Green River, Utah. Ecotoxicol Environ Safe 2000b;45:132–147.

Hamilton SJ, Holley KM, Buhl KJ, Bullard FA, Weston LF, McDonald SF. The evaluation of contaminant impacts on razorback sucker held in flooded bottomland sites near

Grand Junction, Colorado – 1996. Final report, US Geological Survey, Yankton, SD, 2001a, p. 302.

Hamilton SJ, Holley KM, Buhl KJ, Bullard FA, Weston LK, McDonald SF. The evaluation of contaminant impacts on razorback sucker held in flooded bottomland sites near Grand Junction, Colorado – 1997. Final report, US Geological Survey, Yankton, SD, 2001b, p. 229.

Hamilton SJ, Holley KM, Buhl KJ. Hazard assessment of selenium to endangered razorback suckers (*Xyrauchen texanus*). Sci Total Environ 2002a;291:111–121.

Hamilton SJ, Buhl KJ, Lamothe PJ. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: June 2000. Final report as part of the USGS Western US Phosphate Project. US Geological Survey, Yankton, SD, 2002b, p. 70.

Hamilton SJ, Holley KM, Buhl KJ, Bullard FA, Weston LK, McDonald SF. Evaluation of flushing of a backwater channel: concentrations of selenium and other inorganic elements in water, sediment, invertebrates, forage fish, and Colorado pikeminnow. Final report prepared for the Upper Colorado River Endangered Fish Recovery Program. US Geological Survey, Field Research Station, Yankton, SD, 2003, p. 149.

Hansen LD, Maier KJ, Knight AW. The effect of sulfate on the bioconcentration of selenate by *Chironomus decorus* and *Daphnia magna*. Arch Environ Contam Toxicol 1993;25:72–78.

Hansen D, Duda PJ, Zayed A, Terry N. Selenium removal by constructed wetlands: role of biological volatilization. Environ Sci Technol 1998;32:591–597.

Harris T. The selenium question. Defenders March–April 1986;10–20.

Harris T, Morris J, Williamson M. Selenium – toxic trace element threatens the west; the Bee uncovers conspiracy of silence. The Sacramento Bee, Sacramento, CA, 8–10 September, 1985.

Heinz GH. Selenium in birds. In: Beyer WN, Heinz GH, Redmon-Norwood AW, editors. Environmental contaminants in wildlife – interpreting tissue concentrations. Boca Raton, FL: Lewis Publishers, CRC Press, 1996. p. 447–458.

Heinz GH, Fitzgerald MA. Overwinter survival of mallards fed selenium. Arch Environ Contam Toxicol 1993a;25:90–94.

Heinz GH, Fitzgerald MA. Reproduction of mallards following overwinter exposure to selenium. Environ Pollut 1993b;81:117–122.

Heinz GH, Hoffman DJ, Krynitsky AJ, Weller DMG. Reproduction in mallards fed selenium. Environ Toxicol Chem 1987;6:423–433.

Heinz GH, Hoffman DJ, Gold LG. Toxicity of organic and inorganic selenium to mallard ducklings. Arch Environ Contam Toxicol 1988;17:561–568.

Heinz GH, Hoffman DJ, Gold LG. Impaired reproduction of mallards fed an organic form of selenium. J Wildl Manage 1989;53:418–428.

Heinz GH, Hoffman DJ, LeCaptain LJ. Toxicity of seleno-L-methionine, seleno-DL-methionine, high selenium wheat, and selenized yeast to mallard ducklings. Arch Environ Contam Toxicol 1996;30:93–99.

Heinz GH, Hoffman DJ. Methylmercury chloride and selenomethionine interactions on health and reproduction in mallards. Environ Toxicol Chem 1998;17:139–145.

Henderson JD, Maurer TC, Schwarzbach SE. Assessing selenium contamination in two San Joaquin Valley, California sloughs. Draft report to the US Fish and Wildlife Service, Region 1, Division of Environmental Contaminants, Sacramento, CA, 1995, p. 19.

Hermanutz RO. Malformation of the fathead minnow (*Pimephales promelas*) in an ecosystem with elevated selenium concentrations. Bull Environ Contam Toxicol 1992;49:290–294.

Hermanutz RO, Allen KN, Roush TH, Hedtke SF. Effects of elevated selenium concentrations on bluegills (*Lepomis macrochirus*) in outdoor experimental streams. Environ Toxicol Chem 1992;11:217–224.

Hicks BD, Hilton JW, Ferguson HW. Influence of dietary selenium on the occurrence of nephrocalcinosis in the rainbow trout, *Salmo gairdneri* Richardson. J Fish Dis 1984;7:379–389.

Hill CH. Interrelationships of selenium with other trace elements. Fed Am Soc Exp Biol 1975;34:2096–2100.

Hilton JW, Hodson PV. Effect of increased dietary carbohydrate on selenium metabolism and toxicity in rainbow trout (*Salmo gairdneri*). J Nutr 1983;113:1241–1248.

Hilton JW, Hodson PV, Slinger SJ. The requirement and toxicity of selenium in rainbow trout (*Salmo gairdneri*). J Nutr 1980;110:2527–2535.

Hilton JW, Hodson PV, Slinger SJ. Absorption, distribution, half-life and possible routes of elimination of dietary selenium in juvenile rainbow trout (*Salmo gairdneri*). Comp Biochem Physiol C 1982;71:49–55.

Hodson PV, Hilton JW. The nutritional requirements and toxicity to fish of dietary and waterborne selenium. Environ Biogeochem Ecol Bull (Stockholm) 1983;35:335–340.

Hodson PV, Spry DJ, Blunt BR. Effects on rainbow trout (*Salmo gairdneri*) of a chronic exposure to waterborne selenium. Can J Fish Aquat Sci 1980;37:233–240.

Hoffman DJ. Role of selenium toxicity and oxidative stress in aquatic birds. Aquat Toxicol 2002;57:11–26.

Hoffman DJ, Heinz GH. Embryotoxic and teratogenic effects of selenium in the diet of mallards. J Toxicol Environ Health 1988;24:477–490.

Hoffman DJ, Heinz GH. Effects of mercury and selenium on glutathione metabolism and oxidative stress in mallard ducks. Environ Toxicol Chem 1998;17:161–166.

Hoffman DJ, Ohlendorf HM, Aldrich TW. Selenium teratogenesis in natural populations of aquatic birds in Central California. Arch Environ Contam Toxicol 1988;17:519–525.

Hoffman DJ, Heinz GH, Krynitsky AJ. Hepatic glutathione metabolism and lipid peroxidation in response to excess dietary selenomethionine and selenite in mallard ducklings. J Toxicol Environ Health 1989;27:263–271.

Hoffman DJ, Sanderson CJ, LeCaptain LJ, Cromartie E, Pendleton GW. Interactive effects of boron, selenium, and dietary protein on survival, growth, and physiology in mallard ducklings. Arch Environ Contam Toxicol 1991;20:288–294.

Hoffman DJ, Sanderson CJ, LeCaptain LJ, Cromartie E, Pendleton GW. Interactive effects of arsenate, selenium, and dietary protein on survival, growth, and physiology in mallard ducklings. Arch Environ Contam Toxicol 1992a;22:55–62.

Hoffman DJ, Sanderson CJ, LeCaptain LJ, Cromartie E, Pendleton GW. Interactive effects of selenium, methionine, and dietary protein on survival, growth, and physiology in mallard ducklings. Arch Environ Contam Toxicol 1992b;23:163–171.

Hoffman DJ, Heinz GH, LeCaptain LJ, Eisemann JD, Pendleton GW. Toxicity and oxidative stress of different forms of organic selenium and dietary protein in mallard ducklings. Arch Environ Contam Toxicol 1996;31:120–127.

Holland EA. Arsenic and selenium in the water, sediments, and biota near a coal-fired power plant – Belews Lake, North Carolina. Unplublished thesis, University of North Carolina, Chapel Hill, 1979.

Hothem RL, Lonzarich DG, Takekawa JE, Ohlendorf HM. Contaminants in wintering canvasbacks and scaups from San Francisco Bay, California. Environ Monit Assess 1998;50:67–84.

Howell GO, Hill CH. Biological interaction of selenium with other trace elements in chicks. Environ Health Persp 1978;25:147–150.

Hunn JB, Hamilton SJ, Buckler DR. Toxicity of sodium selenite to rainbow trout fry. Water Res 1987;21:233–238.

Ingersoll CG, Dwyer FJ, May TW. Toxicity of inorganic and organic selenium to *Daphnia magna* (Cladocera) and *Chironomus riparius* (Diptera). Environ Toxicol Chem 1990;9:1171–1181.

Johnson TM, Bullen TD, Zawislanski PT. Selenium stable isotope ratios as indicators of sources and cycling of selenium: results from the Northern reach of San Francisco Bay. Environ Sci Technol 2000;34:2075–2079.

Kennedy CJ, McDonald LE, Loveridge R, Strosher MM. The effect of bioaccumulated selenium on mortalities and deformities in the eggs, larvae, and fry of a wild population of cutthroat trout (*Oncorhynchus clarki lewisi*). Arch Environ Contam Toxicol 2000;39:46–52.

Kirkpatrick JS. Selenium levels of water, sediment, and biota of Martin Lake. Austin, TX: Texas Department of Water Resources, 1980.

Klasing KC. Comparative avian nutrition. New York: Oxford University Press, 1998.

Klauda RJ. Acute and chronic effects of waterborne arsenic and selenium on the early life stages of striped bass (*Morone saxatilis*). Report PPRP-98, John Hopkins University, Laurel, MD, 1986.

Klaverkamp JF, Macdonald WA, Lillie WR, Lutz A. Joint toxicity of mercury and selenium in salmonid eggs. Arch Environ Contam Toxicol 1983;12:415–419.

Kleinow KM, Brooks AS. Selenium compounds in the fathead minnow (*Pimephales promelas*) – I. Uptake, distribution, and elimination of orally administered selenate, selenite and l-selenomethionine. Comp Biochem Physiol C 1986;83:61–69.

Klug HL, Petersen DF, Moxon AL. The toxicity of selenium analogues of cystine and methionine. Proc SD Acad Sci 1949;28:117–120.

Klug HL, Lampson GP, Moxon AL. The distribution of selenium and arsenic in the body tissues of rats fed selenium, arsenic, and selenium plus arsenic. Proc SD Acad Sci 1950;29:57–65.

Lemly AD. Response of juvenile centrarchids to sublethal concentrations of waterborne Selenium. I. Uptake, tissue distribution, and retention. Aquat Toxicol 1982;2:235–252.

Lemly AD. Toxicology of selenium in a freshwater reservoir: implications for environmental hazard evaluation and safety. Ecotoxicol Environ Safe 1985;10:314–338.

Lemly AD. Metabolic stress during winter increases the toxicity of selenium to fish. Aquat Toxicol 1993a;27:133–158.

Lemly AD. Teratogenic effects of selenium in natural populations of freshwater fish. Ecotoxicol Environ Safe 1993b;26:181–204.

Lemly AD. Selenium in aquatic organisms. In: Beyer WN, Heinz GH, Redmon-Norwood AW, editors. Environmental contaminants in wildlife – interpreting tissue concentrations. Boca Raton: CRC Press, 1996a. p. 427–445.

Lemly AD. Winter stress syndrome: an important consideration for hazard assessment of aquatic pollutants. Ecotoxicol Environ Safe 1996b;34:223–227.

Lemly AD. Ecosystem recovery following selenium contamination in a freshwater reservoir. Ecotoxicol Environ Safe 1997a;36:275–281.

Lemly AD. Role of season in aquatic hazard assessment. Environ Monit Assess 1997b;45:89–98.

Lemly AD. A teratogenic deformity index for evaluating impacts of selenium on fish populations. Ecotoxicol Environ Safe 1997c;37:259–266.

Lemly AD. Preliminary assessment of selenium hazards on Caribou National Forest, Idaho. US Forest Service, Virginia Tech University, Blacksburg, VA. Report to the US Forest Service, Caribou National Forest, Pocatello, ID, 1999a.

Lemly AD. Selenium impacts on fish: an insidious time bomb. Human Ecol Risk Assess 1999b;5:1139–1151.

Lemly AD. Selenium assessment in aquatic ecosystems – a guide for hazard evaluation and water quality criteria. New York: Springer-Verlag, 2002a.

Lemly AD. Symptoms and implications of selenium toxicity in fish: the Belews Lake case example. Aquat Toxicol 2002b;57:39–49.

Lemly AD, Ohlendorf HM. Regulatory implications of using constructed wetlands to treat selenium-laden wastewater. Ecotoxicol Environ Safe 2002;52:46–56.

Levander OA. Metabolic interrelationships between arsenic and selenium. Environ Health Persp 1977;19:159–164.

Levander OA, Argrett LC. Effects of arsenic, mercury, thallium, and lead on selenium metabolism in rats. Toxicol Appl Pharmacol 1969;14:308–314.

Lindqvist O, Johansson K, Aastrup M, Andersson A, Bringmark L, Hovsenius G, Hakanson L, Iverfeldt A, Meili M, Timm B. Mercury in the Swedish environment – recent research on causes, consequences and corrective methods. Water Air Soil Pollut 1991;55:xi–261.

Linville RG, Luoma SN, Cutter L, Cutter GA. Increased selenium threat as a result of invasion of the exotic bivalve *Potamocorbula amurensis* into the San Francisco Bay-Delta. Aquat Toxicol 2002;57:51–64.

Lorentzen M, Maage A, Julshamn K. Supplementing copper to a fish meal based diet fed to Atlantic salmon parr affects liver copper and selenium concentrations. Aquacult Nutr 1998;4:67–72.

Luoma SN, Linville R. A comparison of selenium and mercury concentrations in transplanted and resident bivalves from north San Francisco Bay. In: 1995 Annual Report San Francisco Estuary Regional Monitoring Program for Trace Substances, 1996, pp. 160–170.

Luoma SN, Presser TS. Forecasting selenium discharges to the San Francisco Bay-Delta Estuary: ecological effects of a proposed San Luis Drain extension. Open-File Report 00-416, US Geological Survey, Menlo Park, CA, 2000.

Maier KJ, Knight AW. The toxicity of waterborne boron to *Daphnia magna* and *Chironomus decorus* and the effects of water hardness and sulfate on boron toxicity. Arch Environ Contam Toxicol 1991;20:282–287.

Maier KJ, Foe CG, Knight AW. Comparative toxicity of selenate, selenite, seleno-DL-methionine, and seleno-DL-cystine to *Daphnia magna*. Environ Toxicol Chem 1993;12:755–763.

Maier KJ, Knight AW. Ecotoxicology of selenium in freshwater systems. Rev Environ Contam Toxicol 1994;134:31–48.

Maier KJ, Foe C, Ogle RS, Williams MJ, Knight AW, Kiffney P, Melton LA. The dynamics of selenium in aquatic ecosystems. In: Proceedings of the University of Missouri's 21st Annual Conference on Trace Substances in Environmental Health. Trace Substances in Environmental Health – XXI, Columbia, MO, 1987, pp. 361–408.

Maier KJ, Ogle RS, Knight AW. The selenium problem in lentic ecosystems. Lake Res Manage 1988;4:155–163.

Maier KJ, Nelson CR, Bailey FC, Klaine SJ, Knight AW. Accumulation of selenium by the aquatic biota of a watershed treated with seleniferous fertilizer. Bull Environ Contam Toxicol 1998;60:409–416.

Malchow DE, Knight AW, Maier KJ. Bioaccumulation and toxicity of selenium in *Chironomus decorus* larvae fed a diet of seleniferous *Selenastrum capricornutum*. Arch Environ Contam Toxicol 1995;29:104–109.

Marier JR, Jaworski JF. Interactions of selenium. National Research Council Canada Report No. 20643, Ottawa, Canada, 1983.

MW (Montgomery Watson). Final – 1998 regional investigation report, southeast Idaho phosphate resource area selenium project. Montgomery Watson, Steamboat Springs, CO, 1999.

MW (Montgomery Watson). 1999 interim investigation data report, southeast Idaho phosphate resource area selenium project. Montgomery Watson, Steamboat Springs, CO, 2000.

Moxon AL. The effect of arsenic on the toxicity of seleniferous grains. Science 1938;88:81.

Naftz DL. Pattern recognition analysis and classification modeling of selenium-producing areas. J Chemometr 1996;10:309–324.

Nakamoto RJ, Hassler TJ. Selenium and other trace elements in bluegills from agricultural return flows in the San Joaquin Valley, California. Arch Environ Contam Toxicol 1992;22:88–98.

NRC (National Research Council). Irrigation-induced water quality problems – what can be learned from the San Joaquin Valley experience. National Research Council, Committee on Irrigation-induced Water Quality Problems. Washington, DC:National Academy Press, 1989.

Nelson SM, Roline RA, Thullen JS, Sartoris JJ, Boutwell JE. Invertebrate assemblages and trace element bioaccumulation associated with constructed wetlands. Wetlands 2000;20:406–415.

Niimi AJ, LaHam QN. Relative toxicity of organic and inorganic compounds of selenium to newly hatched zebrafish (*Brachydanio rerio*). Can J Zool 1976;54:501–509.

Ogle RS, Knight AW. Effects of elevated foodborne selenium on growth and reproduction of the fathead minnow (*Pimephales promelas*). Arch Environ Contam Toxicol 1989;18:795–803.

Ogle RS, Maier KJ, Kiffney P, Williams MJ, Brasher A, Melton LA, Knight AW. Bioaccumulation of selenium in aquatic ecosystems. Lake Reservoir Manage 1988;4:165–173.

Ohlendorf HM. Bioaccumulation and effects of selenium in wildlife. In: Jacobs LW, editor. Selenium in Agriculture and the Environment, Soil Science Society of America Special Publication Number 23, Madison, WI, 1989, pp. 133–177.

Ohlendorf HM. Selenium was a time bomb. Human Ecol Risk Assess 1999;5:1181–1185.

Ohlendorf HM, Gala WR. Selenium and Chevron Richmond Refinery's water enhancement wetland: a response to A.D. Lemly, 1999. Human Ecol Risk Assess 2000;6:903–905.

Ohlendorf HM, Skorupa JP. Selenium in relation to wildlife and agricultural drainage water. In: Proceedings of the 4th International Symposium on Uses of Selenium and Tellurium, Selenium–Tellurium Development Association, Darien, CT, 1989, pp. 314–338.

Ohlendorf HM, Hoffman DJ, Saiki MK, Aldrich TW. Embryonic mortality and abnormalities of aquatic birds: apparent impacts of selenium from irrigation drainwater. Sci Total Environ 1986a;52:49–63.

Ohlendorf HM, Lowe RW, Kelly PR, Harvey TE. Selenium and heavy metals in San Francisco Bay diving ducks. J Wildl Manage 1986b;50:64–71.

Ohlendorf HM, Hothem RL, Aldrich TW. Bioaccumulation of selenium by snakes and frogs in the San Joaquin Valley, California. Copeia 1988;3:704–710.

Ohlendorf HM, Hothem RL, Bunck CM, Marois KC. Bioaccumulation of selenium in birds at Kesterson Reservoir, California. Arch Environ Contam Toxicol 1990;19:495–507.

Ohlendorf HM, Skorupa JP, Saiki MK, Barnum DA. Food-chain transfer of trace elements to wildlife. In: Allen RG, editor. Management of irrigation and drainage systems – integrated perspectives. New York: American Society of Civil Engineers, 1993. p. 596–603.

Osmundson BC, May TW, Osmundson DB. Selenium concentrations in the Colorado pikeminnow (*Ptychocheilus lucius*): relationship with flows in the upper Colorado River. Arch Environ Contam Toxicol 2000;38:479–485.

Paulsson K, Lundbergh K. The selenium method for treatment of lakes for elevated levels of mercury in fish. Sci Total Environ 1989;87–88:495–507.

Paulsson K, Lundbergh K. Treatment of mercury contaminated fish by selenium addition. Water Air Soil Pollut 1991;56:833–841.

Paulsson K, Lundbergh K. Selenium treatment of mercury-contaminated water systems. In: Proceedings of Selenium-Tellurium Development Association's Fifth International Symposium, Brussels, Belgium, 1994, pp. 287–290.

Paveglio FL, Clifton SD. Selenium accumulation and ecology of the San Joaquin kit fox in the Kesterson National Wildlife Refuge Area. US Fish and Wildlife Service, San Luis National Wildlife Refuge, Los Banos, CA. Final report to the US Bureau of Reclamation, 1988.

Pelletier E. Mercury-selenium interactions in aquatic organisms: a review. Mar Environ Res 1985;18:111–132.

Pendergast JF, Ausley LW, Bro-Rasmussen F, Cappel CR, Delos C, Dorward-King EJ, Ethier G, Hansen DJ, LeBlanc NE, Lee CM, Viteri A Jr. Regulatory practice for metals. In: Bergman HL, Dorward-King EJ, editors. Society of Environmental Toxicology and Chemistry Technical Publication Series. Reassessment of metals criteria for aquatic life protection-priorities for research and implementation. SETAC Press, Pensacola, FL, 1997, pp. 13–30.

Piper DZ, Skorupa JP, Presser TS, Hardy MA, Hamilton SJ, Huebner M, Gulbrandsen RA. The Phosphoria Formation at the Hot Springs Mine in southeast Idado: A source of selenium and other trace elements to surface water, ground water, vegetation, and biota. US Geological Survey Open File Report 00-050, Menlo Park, CA, 2000, p. 73.

Presser TS. The Kesterson effect. Environ Manage 1994;18:437–454.

Presser TS, Ohlendorf HM. Biogeochemical cycling of selenium in the San Joaquin Valley, California, USA. Environ Manage 1987;11:805–821.

Presser TS, Piper DZ. Mass balance approach to selenium cycling through the San Joaquin Valley: from source to river to bay. In: Frankenberger Jr WT, Engberg RA, editors. Environmental chemistry of selenium. New York: Marcel Dekker, 1998. p. 153–182.

Presser TS, Sylvester MA, Low WH. Bioaccumulation of selenium from natural geologic sources in Western states and its potential consequences. Environ Manage 1994;18:423–436.

Puls R. Mineral levels in animal health. 2nd ed. Clearbrook, BC, Canada: Sherpa International, 1994.

Pyron M, Beitinger TL. Effect of selenium on reproductive behavior and fry of fathead minnows. Bull Environ Contam Toxicol 1989;42:609–613.

Rapport DJ, Regier HA, Hutchinson TC. Ecosystem behavior under stress. Am Nat 1985;125:617–640.

Rich, 1999 Rich (AA Rich and Associates). FMC phosphate mine expansion. Fishery Resources Technical Report, A.A. Rich and Associates, Fisheries and Ecological Consultants, San Anselmo, CA, 1999.

Riedel GF, Ferrier DP, Sanders JG. Uptake of selenium by freshwater phytoplankton. Water Air Soil Pollut 1991;57–58:23–30.

Rudd JWM, Turner MA, Townsend BE, Swick A, Furutani A. Dynamics of selenium in mercury-contaminated experimental freshwater ecosystems. Can J Fish Aquat Sci 1980;37:848–857.

Saiki MK. A field example of selenium contamination in an aquatic food chain. In: Selenium in the Environment, Publication No. CAT1/860201, California Agricultural Technology Institute, California State University, Fresno, 1986, pp. 67–76.

Saiki MK, Lowe TP. Selenium in aquatic organisms from subsurface agricultural drainage water, San Joaquin Valley, California. Arch Environ Contam Toxicol 1987;16:657–670.

Saiki MK, Ogle RS. Evidence of impaired reproduction by western mosquitofish inhabiting seleniferous agricultural drainwater. Trans Am Fish Soc 1995;124:578–587.

Saiki MK, Schmitt CJ. Population biology of bluegills, *Lepomis macrochirus*, in lotic habitats on the irrigated San Joaquin Valley floor. Calif Fish Game 1985;71:225–244.

Sandholm M, Oksanen HE, Pesonen L. Uptake of selenium by aquatic organisms. Limnol Oceanogr 1973;18:496–499.

Sato T, Ose Y, Sakai T. Toxicological effect of selenium on fish. Environ Pollut 1980;21:217–224.

Schultz R, Hermanutz R. Transfer of toxic concentrations of selenium from parent to progeny in the fathead minnow (*Pimephales promelas*). Bull Environ Contam Toxicol 1990;45:568–573.

Seiler RL. Prediction of areas where irrigation drainage may induce selenium contamination of water. J Environ Qual 1995;24:973–979.

Seiler RL. Synthesis of data from studies by the National Irrigation Water-Quality Program. Water Res Bull 1996;32:1233–1245.

SETAC (Society of Environmental Toxicology and Chemistry). Meeting program: sustaining global environmental integrity. Society of Environmental Toxicology and Chemistry 20th Annual Meeting, Pensacola, FL, 1999.

Skorupa JP. Selenium poisoning of fish and wildlife in nature: lessons from twelve real-world examples. In: Frankenberger

Jr WT, Engberg RA, editors. Environmental chemistry of selenium. New York: Marcel Dekker, 1998. p. 315–354.

Skorupa JP. Beware missing data and undernourished statistical models: comment on Fairbrother et al.'s critical evaluation. Human Ecol Risk Assess 1999;5:1255–1262.

Skorupa JP, Ohlendorf HM. Contaminants in drainage water and avian risk thresholds. In: Dinar A, Zilberman D, editors. The economics and management of water and drainage in agriculture. Boston, MA: Kluwer Academic Publishers, 1991. p. 345–368.

Skorupa JP, Morman SP, Sefchick-Edwards JS. Guidelines for interpreting selenium exposures of biota associated with non-marine aquatic habitats. US Fish and Wildlife Service, Sacramento, CA. Final report to the National Irrigation Water Quality Program, Washington, DC, 1996.

Sorensen EMB. The effects of selenium on freshwater teleosts. In: Hodgson E, editor. Reviews in environmental toxicology 2. New York: Elsevier, 1986. p. 59–116.

Sorensen EMB. Selenium accumulation, reproductive status, and histopathological changes in environmentally exposed redear sunfish. Arch Toxicol 1988;61:324–329.

Sorensen EMB. Metal poisoning in fish. Boca Raton, FL: CRC Press, 1991.

Sorensen EMB, Bauer TL. Hematological dyscrasia in teleosts chronically exposed to selenium-laden effluent. Arch Environ Contam Toxicol 1983;12:135–141.

Sorensen EMB, Bauer TL. A correlation between selenium accumulation in sunfish and changes in condition factor and organ weight. Environ Pollut Ser A 1984;34:357–366.

Sorensen EMB, Bauer TL, Bell JS, Harlan CW. Selenium accumulation and cytotoxicity in teleosts following chronic, environmental exposure. Bull Environ Contam Toxicol 1982;29:688–696.

Sorensen EMB, Bauer TL, Harlan CW, Pradzynski AH, Bell JS. Hepatocyte changes following selenium accumulation in a freshwater teleost. Am J Forensic Med Pathol 1983;4:25–32.

Sorensen EMB, Cumbie PM, Bauer TL, Bell JS, Harlan CW. Histopathological, hematological, condition-factor, and organ weight changes associated with selenium accumulation in fish from Belews Lake, North Carolina. Arch Environ Contam Toxicol 1984;13:153–162.

Spallholz JE, Hoffman DJ. Selenium toxicity: cause and effects in aquatic birds. Aquat Toxicol 2002;57:27–37.

Stanley TR Jr, Spann JW, Smith GJ, Rosscoe R. Main and interactive effects of arsenic and selenium on mallard reproduction and duckling growth and survival. Arch Environ Contam Toxicol 1994;26:444–451.

Stanley TR Jr, Smith GJ, Hoffman DJ, Heinz GH, Rosscoe R. Effects of boron and selenium on mallard reproduction and duckling growth and survival. Environ Toxicol Chem 1996;15:1124–1132.

Stephens D, Waddell B, DuBois K, Peterson E. Field screening of water quality, bottom sediment, and biota associated with the Emery and Scofield Project areas, central Utah, 1994. Water-Resources Investigations Report 96-4298. US Geological Survey, Salt Lake City, UT, 1997.

Stoewsand GS, Bache CA, Lisk DJ. Dietary selenium protection of methylmercury intoxication of Japanese quail. Bull Environ Contam Toxicol 1974;11:152–156.

Terry N, Zayed A. Phytoremediation of selenium. In: Frankenberger Jr WT, Engberg RA, editors. Environmental chemistry of selenium. New York: Marcel Dekker, 1998. p. 633–655.

Thapar NT, Guenthner E, Carlson CW, Olson OE. Dietary selenium and arsenic additions to diets for chickens over a life cycle. Poultry Sci 1969;48:1988–1993.

Turner MA, Rudd JWM. The English-Wabigoon River system: III. Selenium in lake enclosures: its geochemistry, bioaccumulation, and ability to reduce mercury bioaccumulation. Can J Fish Aquat Sci 1983;40:2228–2240.

Turner MA, Swick AL. The English-Wabigoon River system: IV. Interaction between mercury and selenium accumulated from waterborne and dietary sources by northern pike (*Esox lucius*). Can J Fish Aquat Sci 1983;40:2241–2250.

URS. Environmental impact statement and environmental impact report, Volume I-Main text and Volume 2 – Appendices. Report to the US Bureau of Reclamation and San Luis and Delta-Mendota Water Authority, URS, Oakland, CA, 2000.

USDOI (US Department of the Interior). Guidelines for interpretation of the biological effects of selected constituents in biota, water, and sediment. National Irrigation Water Quality Program Information Report No. 3, Denver, CO, 1998.

USEPA (US Environmental Protection Agency). Estimating concern levels for concentrations of chemical substances in the environment. US Environmental Protection Agency, Washington, DC, 1984.

USEPA (US Environmental Protection Agency). Ambient water quality criteria for selenium – 1987. Publication EPA 440/5-87-006, US Environmental Protection Agency, Washington, DC, 1987.

USEPA (US Environmental Protection Agency). Report on the peer consultation workshop on selenium aquatic toxicity and bioaccumulation. Publication EPA 822-R-98-007, US Environmental Protection Agency, Washington, DC, 1998.

USFWS (US Fish and Wildlife Service). Recovery implementation program for endangered fish species in the upper Colorado River basin. US Fish and Wildlife Service, Denver, CO, 1987.

USFWS (US Fish and Wildlife Service). Agricultural irrigation drainwater studies in support of the San Joaquin Valley Drainage Program. US Fish and Wildlife Service, Columbia, MO, Final report to the San Joaquin Valley Drainage Program, Sacramento, CA, 1990.

Vandermeulen JH, Foda A. Cycling of selenite and selenate in marine phytoplankton. Mar Biol 1988;98:115–123.

Vencil B. The Migratory Bird Treaty Act – protecting wildlife on our national refuges – California's Kesterson Reservoir, a case in point. Nat Res J 1986;26:609–627.

Whanger PD. Selenium and heavy metal toxicity. In: Spallholz JE, Martin JL, Ganther HE, editors. Selenium in biology

and medicine. Westport, CT: AVI Publishing, 1981. p. 230–255.

Woock SE. Accumulation of selenium by golden shiners *Notemigonus crysoleucas*. Hyco Reservoir N.C. cage study 1981–1982. Carolina Power and Light Company, New Hill, NC, 1984.

Woock SE, Garrett WR, Partin WE, Bryson WT. Decreased survival and teratogenesis during laboratory selenium exposures to bluegill, *Lepomis macrochirus*. Bull Environ Contam Toxicol 1987;39:998–1005.

Zhang Y, Moore JN. Selenium fractionation and speciation in a wetland system. Environ Sci Technol 1996;30:2613–2619.